MODERN APPROACHES TO THE EARLY DIAGNOSIS AND NEPHROPROTECTION OF DIABETIC KIDNEY DISEASE

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Abstract. Diabetic kidney disease is one of the most common complications of diabetes mellitus and an important cause of chronic kidney disease and cardiovascular morbidity. Its development is associated with chronic hyperglycemia, glomerular hemodynamic changes, oxidative stress, and inflammation. articular attention is given to renin–angiotensin system inhibitors, sodium–glucose cotransporter 2 inhibitors, and other medications with proven renal benefits. Early laboratory diagnosis, regular monitoring, and individualized treatment are essential for preserving kidney function and reducing cardiovascular risk.

Keywords: diabetic kidney disease, diabetes mellitus, albuminuria, estimated glomerular filtration rate, chronic kidney disease, nephroprotection.

Introduction. Diabetic kidney disease is a chronic complication of diabetes mellitus characterized by structural and functional abnormalities of the kidneys. It contributes substantially to the global burden of chronic kidney disease (CKD) and is associated with an increased risk of cardiovascular events, kidney failure, and premature mortality. Early detection is particularly important because appropriate treatment can slow the progression of renal damage [1, 2]. Traditionally, diabetic kidney disease has been associated with persistent albuminuria followed by a decline in glomerular filtration rate (GFR).Some patients develop reduced GFR without significant albuminuria. Assessment of kidney function should not rely on a single laboratory parameter.

Materials and Methods. A narrative review of scientific publications and clinical practice guidelines on diabetic kidney disease was conducted. The review focused on KDIGO recommendations, the joint consensus of the American Diabetes Association and KDIGO, and studies addressing laboratory diagnosis and contemporary treatment strategies [1–4].

Pathogenesis of Diabetic Kidney Disease. The development of diabetic kidney disease involves several interconnected mechanisms, including chronic hyperglycemia, intraglomerular hypertension, activation of the renin–angiotensin–aldosterone system, oxidative stress, and chronic inflammation. Persistent hyperglycemia promotes the formation of advanced glycation end products, which alter the glomerular basement membrane and impair podocyte function. These changes increase glomerular permeability and contribute to albumin leakage into the urine [2, 5]. In the early stages, glomerular hyperfiltration may occur. Although GFR can initially remain normal or elevated, this does not exclude ongoing renal injury. Over time, progressive glomerulosclerosis, tubulointerstitial fibrosis, and vascular changes may lead to a decline in kidney function. Arterial hypertension, obesity, dyslipidemia, and poor glycemic control further increase renal risk.

Laboratory Diagnosis and Assessment of Disease Progression. Early diagnosis is based primarily on two parameters: urine albumin-to-creatinine ratio (UACR) and estimated GFR. According to KDIGO recommendations, these measurements provide complementary information about kidney damage and filtration function and should be assessed together [1].

UACR is commonly measured in a spot urine sample. Albuminuria is classified into three categories:

- A1: less than 30 mg/g creatinine — normal to mildly increased;

- A2: 30–300 mg/g — moderately increased;

- A3: greater than 300 mg/g — severely increased.

Persistent albuminuria is an important marker of kidney damage and is associated with an increased risk of CKD progression and cardiovascular complications. However, albumin excretion may temporarily increase during urinary tract infections, fever, strenuous exercise, or marked hyperglycemia. Therefore, an abnormal result should generally be confirmed by repeat testing [1, 3].

Estimated GFR is usually calculated from serum creatinine using a validated equation. In selected patients, cystatin C and a combined creatinine–cystatin C equation may provide a more accurate estimate, particularly when muscle mass or other factors make creatinine less reliable. Assessment of both UACR and eGFR enables clinicians to classify CKD, evaluate prognosis, and determine the appropriate frequency of monitoring [1]. Laboratory results should be interpreted in conjunction with the patient's clinical history, duration of diabetes, blood pressure, glycemic control, medication use, and associated diseases.

Modern Approaches to Nephroprotection. The main goals of treatment are to preserve kidney function, reduce albuminuria, control cardiovascular risk, and prevent progression to kidney failure. Management includes individualized glycemic control, blood pressure management, a balanced diet, regular physical activity, smoking cessation, and avoidance of unnecessary exposure to nephrotoxic medications [2, 3]. Angiotensin-converting enzyme inhibitors and angiotensin II receptor blockers are important treatment options for patients with diabetes, hypertension, and albuminuria when clinically indicated. These medications reduce intraglomerular pressure and albuminuria and may slow the progression of kidney damage. Blood pressure, serum creatinine, and potassium should be monitored after treatment initiation and dose adjustment [1, 3]. Sodium–glucose cotransporter 2 inhibitors (SGLT2 inhibitors) have become a key component of nephroprotective therapy. Clinical evidence demonstrates that these drugs can reduce the risk of CKD progression and cardiovascular events in

eligible patients. Their use depends on eGFR, comorbidities, contraindications, and current clinical recommendations. An initial decrease in eGFR may occur after initiation and should be interpreted in the clinical context rather than automatically considered evidence of kidney injury [1,3,8].

Finerenone, a nonsteroidal mineralocorticoid receptor antagonist, may be considered in selected patients with type 2 diabetes and persistent albuminuria despite standard treatment. Serum potassium and kidney function must be monitored. Glucagon-like peptide-1 receptor agonists may also be appropriate for certain patients, particularly when additional glycemic, weight-management, or cardiovascular benefits are needed [1, 2, 4].

Monitoring typically includes UACR, eGFR, blood pressure, glycemic parameters, and serum potassium when indicated. Nephrology consultation should be considered in patients with rapid eGFR decline, increasing albuminuria, resistant hypertension, persistent electrolyte disturbances, or suspected kidney disease of another cause [1].

Conclusion. Diabetic kidney disease develops through interacting metabolic, hemodynamic, inflammatory, and structural mechanisms. Early diagnosis requires the combined assessment of albuminuria and estimated GFR, as either parameter alone may fail to identify the full extent of renal risk. Regular laboratory monitoring and individualized management are essential for slowing disease progression and reducing renal and cardiovascular complications.

References

1. Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group. KDIGO 2024 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. Kidney Int. 2024;105(4 Suppl):S117–S314. Doi:10.1016/j.kint.2023.10.018.

2. De Boer IH, Khunti K, Sadusky T, Tuttle KR, Neumiller JJ, Rhee CM, et al. Diabetes management in chronic kidney disease: a consensus report by the American Diabetes Association (ADA) and Kidney Disease: Improving Global Outcomes (KDIGO). Diabetes Care. 2022;45(12):3075–3090. Doi:10.2337/dci22-0027.
3. Kidney Disease: Improving Global Outcomes (KDIGO) Diabetes Work Group. KDIGO 2022 Clinical Practice Guideline for Diabetes Management in Chronic Kidney Disease. Kidney Int. 2022;102(5 Suppl):S1–S127. Doi:10.1016/j.kint.2022.06.008.
4. American Diabetes Association Professional Practice Committee. Chronic Kidney Disease and Risk Management: Standards of Care in Diabetes—2026. Diabetes Care. 2026;49(Suppl 1).
5. Gaddy G, Elrggal M, Madariga H, Kelly A, Colbert GB, Lerma E. Diabetic kidney disease. Dis Mon. 2025;71(4):101848. Doi:10.1016/j.disamonth.2024.101848.
6. Yusupova, S., & Delkasheva, S. (2026). CHRONIC OBSTRUCTIVE PULMONARY DISEASE WITH OBESITY COMORBIDITY: RENAL DYSFUNCTION AND OPTIMIZATION OF TREATMENT. JANUBIY OROL BO'YI TIBBIYOT JURNALI, 2(Maxsusu son), 596–599. https://doi.org/10.5281/zenodo.19807327
7. Rout P, Jialal I. Diabetic nephropathy. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025.
8. Делкашева Ш. Д. АНЕМИЯ И САХАРНЫЙ ДИАБЕТ: ПРИЧИНЫ ВОЗНИКНОВЕНИЯ И ПРИНЦИПЫ ЛЕЧЕНИЯ // Экономика и социум. 2022. №2-2 (93). URL: https://cyberleninka.ru/article/n/anemiya-i-saharnyy-diabet-prichiny-vozniknoveniya-i-printsipy-lecheniya (дата обращения: 04.11.2026).