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Novel Biomarkers for Early Detection and Prognosis of Diabetic Kidney Disease

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ABSTRACT

Diabetic kidney disease (DKD) is a common microvascular complication of diabetes mellitus and a major cause of chronic kidney disease (CKD) and end-stage kidney disease (ESKD) in the world. While traditional markers, such as urinary albumin-to-creatinine ratio, serum creatinine, and estimated glomerular filtration rate remain pivotal in clinical evaluation, they often fall short in identifying subclinical renal damage and in predicting the trajectory of a particular disease. This study assesses newer markers of early detection, prognostic stratification and therapeutic monitoring of DKD. It covers various biomarkers of glomerular and tubular injury, inflammation, oxidative stress, fibrosis and metabolic dysregulation such as nephrin, podocin, kidney injury molecule-1, neutrophil gelatinase-associated lipocalin, tumour necrosis factor receptors, CCL20, trimethylamine N-oxide and fibrosis-related mediators. The study also discusses genomic, epigenomic, transcriptomic, proteomic, and metabolomic methods, highlighting the potential of combination of multi-omics, systems biology, artificial intelligence, and machine learning to enhance personalized risk prediction. The use of a panel of biomarkers including molecular, clinical, imaging, and digital more accurate for diagnosis and prognosis than a single-marker approach. But challenges of external validation, assay variability, high assay costs, regulatory demands and the ability to integrate advanced testing into routine workflows have limited clinical translation. Thus, large-scale prospective studies, common analytical procedures and cost-effectiveness analyses are needed. The use of validated molecular markers in conjunction with current tools leads to earlier intervention, tailored treatment choices, more effective therapeutic monitoring, and better long-term renal and cardiovascular outcomes for DKD.

Keywords: Diabetic kidney disease; Early detection; Novel biomarkers; Multi-omics; Precision nephrology

1. Introduction

Diabetic kidney disease (DKD) is the most common and most serious microvascular complication of diabetes mellitus and is a major cause of chronic kidney disease (CKD) and end-stage kidney disease (ESKD) worldwide. The increased burden of type 1 and 2 diabetes have led to a significant increase in the burden of DKD, with a large burden on health systems because people with DKD require long-term medical care, dialysis and kidney transplantation. Apart from the renal complications, DKD is also a significant public health problem as it is strongly associated with cardiovascular disease, hospitalization and early death. Even with the recent progress made in diabetes treatment and nephroprotective medications, there remains a significant number of diabetic patients who develop progressive renal decline, which demonstrates the need to find better ways to detect and treat diabetes early on (Alicic, Rooney, and Tuttle 2017; de Boer et al. 2020).

DKD pathogenesis is multifactorial and the complexity of the interaction between the metabolic, hemodynamic, inflammatory and fibrotic mechanisms. Hyperglycemia leads to continuous oxidative stress, advanced glycation

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end product production, mitochondrial dysfunction, and activation of inflammatory signaling pathways that all lead to glomerular and tubular injury. While traditionally, DKD has been regarded as a glomerular disease in which the hallmarks are albuminuria, there is increasing evidence that tubular injury, endothelial dysfunction, and interstitial fibrosis are equally important and occur early in the pathogenesis of the disease. The combined effects of structural changes including thickening of the GBM, expansion of the mesangial area, loss of podocytes and tubular damage result in progressive loss of nephrons and loss of GFR. In addition, there is a shift from DHD as a solely glomerular disease to a more comprehensive renal disease involving multiple compartments of the nephron (proximal tubule) in understanding the disease process (De Boer et al. 2022; Gilbert 2017; Sugahara et al. 2021). Renal damage occurs long before clinical signs can be detected, making early diagnosis of DKD critical. Current diagnostic markers, such as urinary albumin excretion and estimated glomerular filtration rate (eGFR), continue to be the main tools for clinical evaluation, but are not particularly sensitive to subclinical kidney injury. Some patients develop progressive renal impairment despite normal albumin excretion while others have albuminuria in the short term that is not necessarily indicative of future renal disease. Therefore, a late diagnosis often leads to permanent destruction of the nephrons and loss of therapeutic possibilities. Improved early prognostic evaluation allows us the opportunity to intervene at the right time with renoprotective treatments, fine-tune glycemic and blood pressure control, and start new treatments that are emerging which slow disease progression. In recent years, clinical advances, including introduction of mineralocorticoid receptor antagonists like finerenone in high-risk patients who are well selected for benefit, underscore the need for accurate risk stratification (Bakris et al. 2020; Coca et al. 2017).

Because of the shortcomings of the current clinical markers, there has been a considerable amount of research into novel markers that can detect kidney injury before it is too late for irreversible damage to have taken place. Diagnostic biomarkers for glomerular injury, tubule dysfunction, inflammation, fibrosis, oxidative stress, endothelial damage, and mechanisms of cellular repair give a good insight into the biological and molecular mechanisms involved in the progression of DKD. A wide range of novel candidate biomarkers have emerged from the fields of molecular biology, proteomics, metabolomics, and extracellular vesicle studies that used in diagnosis and prognosis. Furthermore, combining several biomarkers enhance the ability to predict the disease, as multiple mechanisms involved in progression of DKD and the combination of several biomarkers captures the diversity of these mechanisms, which would facilitate personalized risk assessment and therapeutic decisions (Colhoun and Marcovecchio 2018; Kharbanda 2020).

The objective of this study is to give a broad overview of the status quo in novel biomarkers for early detection and prognosis of DKD. It provides a valuable overview of recent developments in molecular and omics-based biomarkers with their biological relevance and clinical implementation and considers the possibility of using these in routine nephrology care. In addition, the study summarizes current evidence from international consensus statements and clinical practice guidelines to reveal emerging therapeutic implications, recent guideline recommendations and future directions for the use of biomarkers in precision medicine for the management of DKD.

2. Current Diagnostic Approaches and Their Limitations

2.1 Conventional Diagnostic Markers (Albuminuria, eGFR, and Serum Creatinine)

Traditionally, the diagnosis and monitoring of diabetic kidney disease (DKD) has been based on three main clinical parameters: urinary albumin excretion, estimated glomerular filtration rate (eGFR), and serum creatinine. Urinary albumin-to-creatinine ratio (UACR) is a well-established surrogate of glomerular injury that is commonly used for the screening of DKD. EGFR and serum creatinine, similarly, are indicators of renal function and are used frequently to stage the disease and assess the progression of chronic kidney disease. These parameters are still the mainstays of contemporary clinical management but mainly reflect function rather than molecular and cellular changes within the kidney. Therefore, these markers are not very sensitive in detecting initial renal damage (Barutta et al. 2021; Mortaji and Wagner 2020). The merits and drawbacks of the techniques currently available for the diagnosis of DKD are summarized in Table 1.

Table 1. Conventional Diagnostic Approaches for Diabetic Kidney Disease and Their Clinical Limitations

Diagnostic Method	Primary Clinical Purpose	Major Advantages	Key Limitations	References
Urinary Albumin-to-Creatinine Ratio (UACR)	Detect albuminuria	Simple, inexpensive, guideline recommended	Poor sensitivity for early tubular injury; normoalbuminuric DKD exists	(Barutta et al. 2021; Persson and Rossing 2018)
Serum Creatinine	Estimate renal function	Widely available	Changes occur only after significant nephron loss	(Mortaji and Wagner 2020)
eGFR	CKD staging	Standard clinical indicator	Poor early detection capability	(Griffin 2022)
Kidney Ultrasound	Structural assessment	Non-invasive	Cannot detect early molecular injury	(Esposito et al. 2023)
Kidney Biopsy	Histopathological confirmation	Gold standard	Invasive; unsuitable for routine monitoring	(Griffin 2022)

2.2 Imaging and Histopathological Assessment

Both ultrasonography and computed tomography (CT) and magnetic resonance imaging (MRI) are useful in determining kidney size and size of any abnormalities, and to rule out other renal diseases. Most of these methods, however, are insensitive to detect subtle microvascular, inflammatory or fibrotic changes typical of early DKD. Renal biopsy is the gold standard for definitive histopathological evaluation, which allows direct visualisation of: glomerular basement membrane thickening, mesangial expansion, podocyte injury, tubular atrophy and interstitial fibrosis. However, biopsy is not suitable for routine screening or serial monitoring, it can be invasive with risks of bleeding and other complications. Therefore, imaging and histopathology are usually reserved for atypical presentations of disease or for when other causes of the disease are suspected but not for the early detection of diabetic kidney injury (Esposito et al. 2023; Griffin 2022).

2.3 Limitations in Early Detection

While the traditional diagnosis methods have helped to manage the care of DKD, there are still many challenges in detecting DKD at an early stage and potentially reversible stage. There is a significant percentage of patients who develop progressive kidney disease with normoalbuminuria and others who develop transient albuminuria who don't continue to deteriorate. The serum creatinine and eGFR may only change following substantial loss of nephrons, which limits the possibilities of intervening in a timely fashion. In addition, these traditional markers offer little information on the intricate mechanisms involved in disease development such as inflammation, oxidative stress, tubular damage, endothelial dysfunction, and fibrosis. There is emerging evidence that molecular mediators are involved in renal injury at a great deal earlier stage than when urine albumin levels or eGFR get measurable (Lousa et al. 2022; Sharma et al. 2023).

2.4 Need for Sensitive and Predictive Biomarkers

These limitations have driven the need for more innovative diagnostic strategies that will improve the detection of kidney damage at an earlier stage, the accuracy of disease prediction and the precision of personalized therapeutic management. In recent years, molecular medicine has led to the discovery of novel biomarkers that correlate with tubular damage, activation of inflammation, fibrosis, and metabolic deregulation, several of which have a better predictive value than conventional clinical markers. Other biomarkers, including Klotho, fibroblast growth factor-23 (FGF23), TNF receptors, and other cardiorenal biomarkers, have been found to have potential for better risk stratification and predicting cardiovascular and renal outcomes in diabetes patients. Meanwhile, the rise of precision medicine and newer therapeutic approaches, such as sodium-glucose cotransporter-2 (SGLT2) inhibitors, have reinforced the importance of having robust biomarkers to identify patient subsets that are most likely to respond to targeted treatment and to enable personalized disease management (Heerspink, Langkilde, and Wheeler 2021; Liu et al. 2021; Mendonça 2025). Figure 1 depicts the shift from outdated methods of diagnosis to molecular biomarker-based diagnosis.

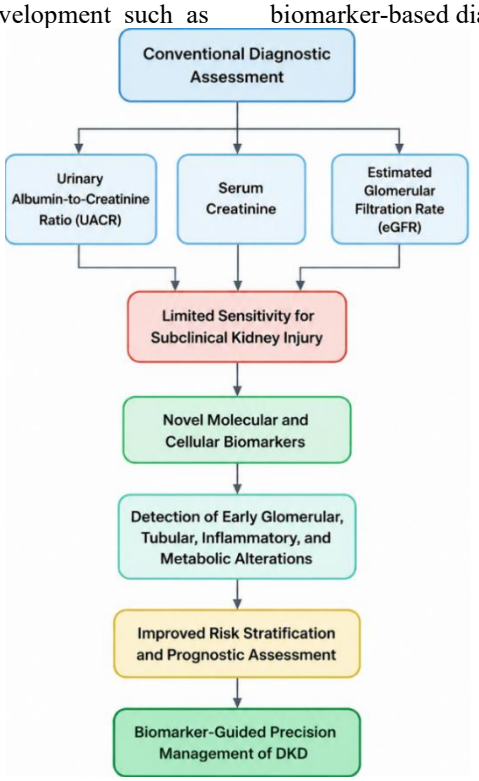

Figure 1. Evolution of Diagnostic Strategies in Diabetic Kidney Disease

3. Emerging Biomarkers for Early Detection of Diabetic Kidney Disease

3.1 Glomerular Injury Biomarkers

One of the earliest pathological events in diabetic kidney disease (DKD) is glomerular injury, which may not be detected clinically until renal function is affected. The traditional measure of glomerular dysfunction is albuminuria, but it is not sensitive enough to diagnose early glomerular changes. Therefore, the search has been turned to identify biomarkers that directly relate to podocyte damage, disruption of the glomerular basement membrane and endothelial cell dysfunction. New data indicates that a combination of circulating and urinary biomarkers with traditional clinical parameters increases diagnostic accuracy and can aid in the earlier detection of disease progression. Additionally, progress in biomarker profiling is improving molecular-level understanding of glomerular disease, and thus facilitating more personalized diagnostic approaches (Persson and Rossing 2018; Przezak et al. 2026).

3.2 Tubular Injury Biomarkers

There is now growing evidence that tubular injury occurs not only as a secondary effect of glomerular dysfunction but is also an early and independent factor contributing to DKD progression. Kidney Injury Molecule-1 (KIM-1) is one of the most well-studied tubular biomarkers due to its strong association with proximal tubular epithelial cell damage. High urinary or plasma KIM-1 levels have been associated with falling renal function, disease severity and poor renal outcomes, indicating that KIM-1 used as a tool to detect renal injury prior to the development of a significant decrease in estimated glomerular filtration rate (eGFR). Other tubular injury markers such as chemokines that mediate renal repair and inflammation show promise as novel predictors of future kidney outcomes and as risk stratification tools (Anandkumar et al. 2023; Massoth et al. 2023).

3.3 Inflammatory Biomarkers

Persistent low-level inflammation is now known to be a key mechanism contributing to the initiation and progression of DKD. Endothelial dysfunction, recruitment of leukocytes, accumulation of extracellular matrix and progressive nephron loss occur due to persistent activation of immune pathways. CC motif chemokines have been particularly interesting and have demonstrated great promise as markers of ongoing renal inflammatory activity among the emerging inflammatory markers. For instance, the concentrations of urinary and circulating CCL20 have been correlated with renal injury and disease severity and useful for diagnostic and prognostic purposes. Because inflammatory processes are generally a precursor to irreversible structural changes, these biomarkers help detect disease activity earlier than traditional clinical measures and potentially identify patients who benefit from targeted anti-inflammatory interventions (Molina-Cazallas et al. 2025). The main pathological pathways involved in the release of biomarkers in DKD are shown in Figure 2.

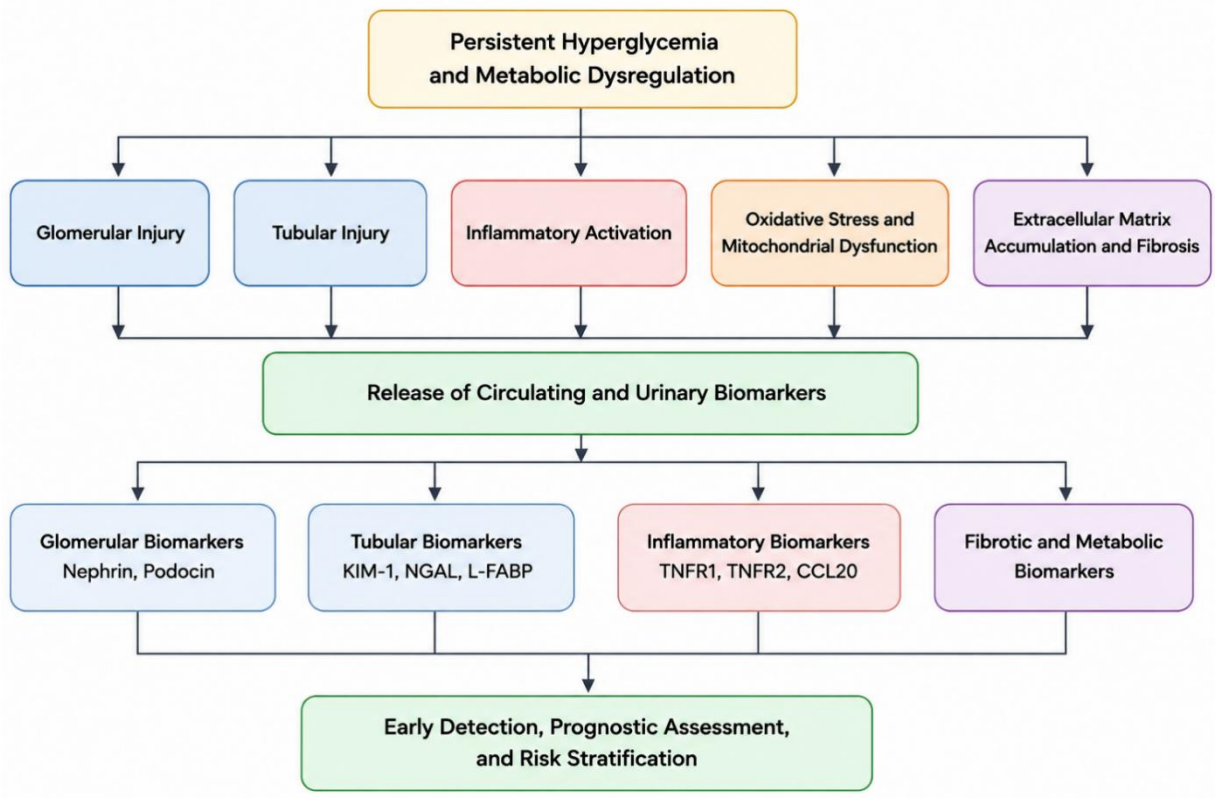

Figure 2. Pathophysiological Sources of Novel Biomarkers in Diabetic Kidney Disease

3.4 Oxidative Stress Biomarkers

Oxidative stress is the key mechanism in hyperglycaemia-induced renal damage. Overproduction of reactive oxygen species leads to damage of glomerular and tubular cells, to alterations of mitochondrial function and to inflammatory signaling pathways that accelerate the progression of the disease. While no single oxidative stress marker is yet in common use in clinical practice,

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there are several potential markers of cell oxidative damage that are being studied as screening markers. Their association with inflammatory and tubular injury markers offers a more comprehensive evaluation of renal injury, enabling clinicians to detect high-risk individuals before the irreversible loss of nephrons (Xing et al. 2024).

3.5 Fibrosis-Related Biomarkers

No matter what insult it is, the last common pathway towards chronic kidney failure is renal fibrosis. The progressive deposition of extracellular matrix, the activation of fibroblasts and tubular atrophy are directly responsible for the irreversible loss of renal function. However, with the advent of systems biology and dynamic multi-omics analyses, new molecular networks have been discovered through which fibrosis progresses, offering potential to detect fibrosis early before the onset of extensive structural damage. Mechanistic modelling and the discovery of biomarkers have enhanced understanding of the complex signalling pathways underlying renal fibrosis and help in developing biomarker panels to monitor disease progression and therapeutic response (Tuechler et al. 2025).

3.6 Metabolic Biomarkers

Metabolites considered potential biomarkers for the early diagnosis and prognosis of DKD, and metabolic dysregulation is important in the pathogenesis of the disease. This relationship between metabolic changes and kidney disease is exemplified by the fact that a compound produced by the gut microbiota called trimethylamine N-oxide (TMAO) is linked to cardiovascular complications and worsening renal function among people with diabetes. In addition, with the advent of multi-omics technologies and artificial intelligence, genomic, transcriptomic, proteomic, and metabolomic data can be combined to discover more sensitive biomarker signatures that can predict disease onset, progression, and treatment response. These precision medicine strategies are poised to revolutionize the future of DKD diagnostics through personalized risk stratification and therapeutic interventions (Li et al. 2026; Winther et al. 2019). The major biomarkers that are being studied for early detection of DKD are outlined in Table 2.

Table 2. Major Emerging Biomarkers for Early Detection of Diabetic Kidney Disease

Biomarker Category	Representative Biomarkers	Biological Significance	Clinical Utility	References
Glomerular	Nephrin, Podocin	Podocyte injury	Early glomerular damage	(Persson and Rossing 2018)
Tubular	KIM-1, NGAL	Tubular epithelial injury	Early tubular damage	(Anandkumar et al. 2023)
Inflammatory	TNFR1, TNFR2, CCL20	Chronic inflammation	Disease progression	(Lousa et al. 2022)
Fibrosis	TGF-β, Collagen biomarkers	Fibrogenesis	Progression monitoring	(Tuechler et al. 2025)
Metabolic	TMAO	Metabolic dysfunction	Cardiovascular and renal risk	(Winther et al. 2019)
Multi-omics	Integrated signatures	Systems biology	Precision diagnosis	(Li et al. 2026)

4. Novel Omics-Based Biomarkers and Advanced Molecular Technologies

4.1 Genomic Biomarkers

Genomic biomarkers have been developed as potential tools to identify people who are genetically at risk for diabetic kidney disease (DKD) before they experience symptoms. Susceptibility genes that predispose to inflammation, oxidative stress, extracellular matrix remodeling and renal fibrosis have been identified by genome-wide association studies and next-generation sequencing, which provide insights into the molecular mechanisms driving the initiation and progression of disease. Genomic biomarkers can be used to identify inherited risk profiles and to support precision medicine approaches that can include individualized prevention and treatment, whereas conventional biomarkers have been used to identify established tissue injury. The increasing prevalence of chronic kidney disease (CKD) globally highlights the need for incorporating genomic data into standard clinical risk stratification systems to enhance patient outcomes in the long run (Meng et al. 2025; Saran et al. 2017).

4.2 Epigenomic Biomarkers

Epigenetic modifications constitute dynamic molecular modifications that impact gene expression without altering the DNA sequence. DKD causes a phenomenon known as “metabolic memory” which is characterized by persistent hyperglycemia leading to DNA methylation, histone modifications and chromatin remodeling, resulting in ongoing renal injury despite improved glycemic control. These epigenetic modifications affect inflammatory pathways, the response to oxidative stress and fibrotic pathways that are involved in the progressive decline of renal function. As a result, epigenomic markers have become of great interest as potential markers of early disease activity and therapeutic response. Additionally, targeting epigenetic mechanisms is considered a potential new approach to slowing CKD progression and to prevent irreversible renal damage (Panizo et al. 2021; Ruiz-Ortega et al. 2020).

4.3 Transcriptomic Biomarkers (mRNA, microRNA, and lncRNA)

Transcriptomic profiling has significantly increased the knowledge of molecular changes that take place during the progression of DKD. Messenger RNA (mRNA), microRNA (miRNA) and long non-coding RNA

(lncRNA) are known to regulate several biological processes such as inflammation, apoptosis, angiogenesis, fibrosis and cell metabolism. These RNA molecules are also promising candidates for early diagnosis and prognosis because altered expression patterns can be detected in blood, urine and renal tissue before development of conventional clinical abnormalities. They reflect constant molecular activity and thus give clues about the severity of the disease and response to therapy. Combining transcriptomic signatures with clinical data is expected to enhance patient stratification and guide personalized management strategies (Meng et al. 2025; Ruiz-Ortega et al. 2020).

4.4 Proteomic Biomarkers

Proteomic technologies allow the characterization of protein expression profiles from the overall protein level in renal injury and have greatly facilitated the discovery of biomarkers in DKD. Many proteins linked to glomerular injury, tubular dysfunction and inflammation, as well as extracellular matrix remodeling, have been identified by urinary and plasma proteomic analysis. Protein markers are especially appealing as they are directly related to the dynamic biological processes and also present a higher specificity when compared to the classic clinical markers. Urinary protein panels have been increasingly recognized for their potential in early diagnosis, disease monitoring, and predicting treatment response through systematic evaluations. Furthermore, the improvement of imaging techniques, such as magnetic resonance imaging (MRI), has complemented proteomic analyses and offers structural and functional information, allowing a complete evaluation of the kidney (Sauriasari, Safitri, and Azmi 2021; Selby and Francis 2025).

4.5 Metabolomic Biomarkers

Metabolomics is the study of small-molecule metabolites that are the products of cellular metabolism and will directly reflect physiological or pathological processes. Amino acid, lipid, glucose metabolism and mitochondrial function are all metabolically disturbed in DKD and possibly before the onset of any renal function changes. These metabolomic signatures present a valuable opportunity for identifying patients with a higher risk of rapid disease progression as well as for gaining insight into the efficacy of therapy. The significance of metabolomic biomarkers in precision nephrology and personalized disease management is growing with the advancement of analytical technologies (Rossiter et al. 2024).

4.6 Multi-Omics Integration and Systems Biology

While single omics platforms offer useful molecular information, combined analysis of genomics, epigenomics, transcriptomics, proteomics, and metabolomics helps to gain a more comprehensive understanding of DKD pathogenesis. The characteristics and clinical applications of major omics technologies are summarized in Table 3.

Table 3. Omics Technologies Applied in Diabetic Kidney Disease

Omics Technology	Major Targets	Clinical Application	Advantages	References
Genomics	DNA variants	Risk prediction	Stable genetic information	(Meng et al. 2025)
Epigenomics	DNA methylation	Disease susceptibility	Detects metabolic memory	(Ruiz-Ortega et al. 2020)
Transcriptomics	mRNA, miRNA, lncRNA	Early diagnosis	Reflects gene expression	(Meng et al. 2025)
Proteomics	Urinary proteins	Disease monitoring	High biological specificity	(Sauriasari et al. 2021)
Metabolomics	Small metabolites	Risk prediction	Functional metabolic profiling	(Rossiter et al. 2024)
Multi-omics	Integrated datasets	Precision medicine	Highest predictive accuracy	(Meng et al. 2025)

Systems biology strategies integrate these multi-dimensional datasets with clinical characteristics, digital phenotyping, imaging and artificial intelligence to discover powerful biomarker signatures that better predict the onset, progression and response to treatment for diseases than individual biomarkers alone. These multimodal approaches facilitate personalized risk prediction, timely interventions, and improved therapeutic decisions, thereby contributing to the field of precision medicine (Li et al. 2026). The use of multi-omics integration is a significant leap towards making biomarker-driven management a regular practice in clinical care, especially as CKD places a heavy burden on healthcare systems in many countries. Multiple omics platforms for precision nephrology are depicted in Figure 3.

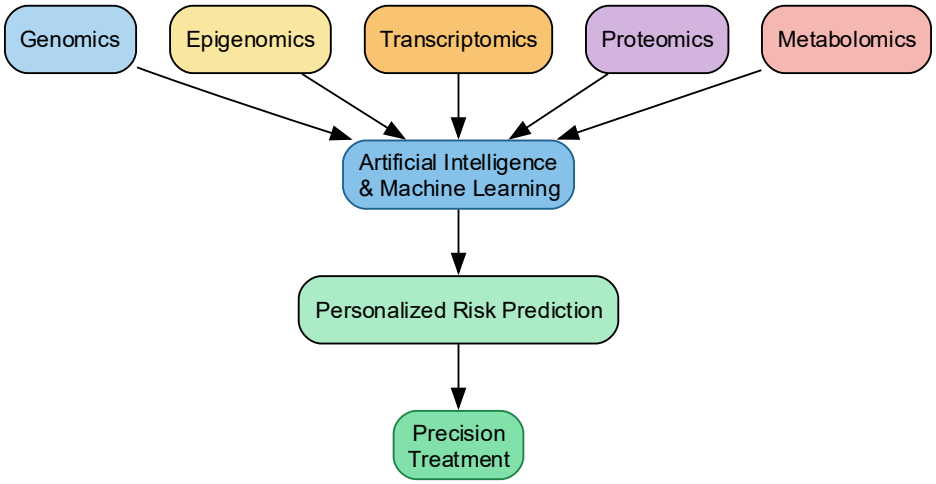

Figure 3. Multi-Omics Framework for Precision Nephrology

5. Biomarkers for Disease Progression, Prognosis, and Therapeutic Monitoring

5.1 Prediction of DKD Progression

The prediction of the progression of diabetic kidney disease (DKD) is still a major clinical challenge, as there is also a significant amount of variation in the timing of onset, the rate of progression, and the response to treatment in patients with DKD. Traditional risk factors, like albuminuria and estimated glomerular filtration rate (eGFR), serve as valuable clinical tools, but they are often unavailable until substantial nephron loss has already taken place. New insights in bioinformatics and next-generation sequencing have allowed the discovery of novel molecular signatures that are associated with progressive renal injury and are more predictive than traditional biomarkers alone. These molecular profiles help to identify high-risk patients earlier and allow timely intervention and individualized disease management (Vastrad and Vastrad 2023; Webster et al. 2017).

5.2 Biomarkers Associated with Renal Function Decline

The progressive loss of kidney function is due to the consequences of glomerular damage, tubular dysfunction, chronic inflammation, oxidative stress, and metabolic dysfunction. Endogenous adenine is one of the recent biomarkers identified that serves as an indicator of kidney failure and death in diabetic patients, suggesting previously ignored metabolic pathways that play a role in the development of DKD. Similarly, changes in gut microbes and their products have been linked to chronic inflammation and immune dysfunction, and to renal fibrosis, and therefore have the promise of serving as non-invasive markers in disease monitoring. These findings expand the knowledge on DKD beyond the classical renal markers and highlight the role of systemic metabolic interactions in the process of renal function loss (Liu et al. 2025; Sharma et al. 2023).

5.3 Risk Stratification for End-Stage Kidney Disease

Identifying patients at risk for end-stage kidney disease (ESKD) early is critical to best manage their clinical care and improve morbidity and mortality outcomes. These molecular markers of inflammation, fibrosis, mineral metabolism and tissue injury, when combined with traditional clinical parameters, offer a more detailed picture of the trajectory of the disease, known as biomarker-based risk stratification. Biomarkers of chronic kidney disease mineral and bone disorder (CKD-MBD), such as those of calcium-phosphate homeostasis, have proven useful for prognosis of long-term renal outcomes and systemic complications. Given the high burden of CKD in the world, and its growing incidence in people with diabetes, the incorporation of predictive markers into clinical practice aids in prioritizing patients for care, optimizing monitoring and resource allocation (Bikbov et al. 2020; Hasparyk et al. 2022).

5.4 Biomarkers for Treatment Response and Personalized Medicine

The increasing focus on precision medicine has led to the rapid evolution of biomarkers that direct personalized therapy and predict response to treatment. Specific pathways are detected by biomarkers allowing to choose therapies based on biological characteristics, not just on clinical parameters. Sodium-glucose cotransporter-2 (SGLT2) inhibitors have been shown to have significant renal and cardiovascular benefits in DKD, and patient selection based on biomarkers could potentially further enhance this therapeutic effect. Moreover, extracellular vesicles (ECsVs), specifically exosomes, offer potential as carriers of molecules (including proteins, nucleic acids, and signaling molecules), which may be of interest in nephrology not only as diagnostic markers but also as therapeutic targets and drug transporters, thus opening new fields for personalized nephrology (Gamez et al. 2025; Vallon and Verma 2021).

5.5 Prognostic Biomarker Panels

However, rather than using individual biomarkers, recent studies are increasingly focusing on the use of multimarker models that combine biochemical, molecular, imaging, and clinical factors for improved predictive power. These types of composite models reflect the multifactorial nature of DKD progression and enhance risk predictions for the development of kidney failure,

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cardiovascular events and death. These integrated prognostic systems are being facilitated by advances in systems biology, machine learning and precision medicine, and can potentially allow for the continuous monitoring of patients and dynamic risk assessment over the course of the disease. Additionally, the overall epidemiological knowledge of kidney disease highlights the need for effective prognosis biomarker panels for better long-term outcomes and to minimize the global burden of renal complications (Bikbov et al. 2020; Cerda et al. 2026).

6. Clinical Translation and Challenges in Biomarker Implementation

6.1 Clinical Validation and Standardization

Clinical validation and standardization is performed in the following way: While many biomarkers are important in the diagnosis and prognosis of diabetic kidney disease (DKD), relatively few have leaped into clinical use. All biomarkers need to be carefully analytically and clinically validated in different patient cohorts before widespread use to demonstrate long-term predictive performance and reproducibility, sensitivity and specificity. The harmonization of methodology, specimen collection, reference values and interpretation values of laboratory tests is also essential for consistency between health care institutions. Recent reviews highlight the need for multi-centre validation of urinary biomarkers and molecular signatures extracted from multi-omics to be incorporated into evidence-based clinical guidelines. In addition, the translation of biomarker discoveries into standardized diagnostic assays is a key pre-step to enhance patient outcomes (Gembillo et al. 2026; Rroji et al. 2026).

6.2 Diagnostic Accuracy and Cost-Effectiveness

The clinical usefulness of any biomarker is not only related to its diagnostic value but also to its cost and availability. The advanced molecular methods including proteomics, metabolomics, transcriptomics and genomic sequencing are more sensitive in identifying early kidney injury, but are complex, expensive, and will typically need special lab facilities, skilled technicians and a significant financial investment. Therefore, it is important to perform cost-effectiveness analyses before the introduction of these technologies into clinical practice. Novel biomarkers could be used in conjunction with existing clinical markers to deliver an ideal balance of diagnostic accuracy and healthcare affordability. Furthermore, molecular biomarkers indicating underlying pathogenic pathways such as impaired endogenous adenine metabolism or mitochondrial dysfunction enable more effective patient stratification for personalized treatment approaches, reducing the need for unnecessary healthcare costs (Hughes et al. 2025; Sharma et al. 2023).

6.3 Regulatory Considerations

In order to be clinically translated, biomarker-based diagnostics must meet regulatory requirements that are far from trivial in terms of analytical validity, clinical performance, safety and quality assurance. The number of regulatory requirements for thorough evidence of clinical utility before approving the use of new biomarkers is growing. At the same time, new internationally generated clinical practice guidelines have started to include precision medicine concepts and molecular biomarkers in the management of chronic kidney disease, expressing a greater degree of confidence in their clinical relevance. But, ongoing assessment in the form of prospective clinical trials and post-marketing surveillance is essential to assure their sustained efficacy and safety in various healthcare environments (Disease and Group 2026).

6.4 Integration into Clinical Workflows

For biomarkers to be adopted into standard nephrology care, they must be easily integrated into current workflows and electronic health record (EHR) systems. Artificial Intelligence or Artificial Intelligence (AI) and Machine Learning (ML) have become important methods for analyzing such large-scale clinical and molecular datasets which will help to automate the risk prediction and provide personalized clinical decision support. Explainable machine learning models using EMR have shown great promise in the detection of high-risk CKD patients and early intervention. In addition, the integration of biomarker information with digital health technologies holds potential to enhance monitoring of diseases, optimize therapeutic decisions, and decrease workload for health care professionals while increasing patient-centered care (Jiang et al. 2026; Martinez Leon, Hilburg, and Susztak 2026).

6.5 Challenges in Routine Clinical Adoption

While much progress has been made in the field of biomarker discovery, there are still some challenges that have prevented the routine use of these in the clinic. These include availability of standardized assays, reliability of the assays in different populations, cost of testing, lack of familiarity of clinicians with testing and uncertainty in reimbursement policies. Furthermore, the implementation of biomarker-guided precision medicine into standard diabetes care will demand a multidisciplinary effort from nephrologists, endocrinologists, clinical labs, bioinformaticians and the regulatory agencies. The path for turning biomarker discoveries into practice is shown in Figure 4.

Figure 4. Clinical Translation Pathway of Biomarkers into Routine Practice

Now, with disease-modifying therapies like sodium-glucose cotransporter-2 (SGLT2) inhibitors becoming more common, it has become even more important to identify reliable biomarkers that can inform a personalized selection of therapies and track therapy response. With the continued development of personalized glycemic management and kidney-protective therapies, biomarker-driven clinical decision-making is likely to be a key part of future DKD management (Klein et al. 2025; Wheeler et al. 2021).

7. Future Perspectives

The future of the diagnosis of diabetic kidney disease (DKD) is attuned to the integration of cutting-edge molecular innovations, artificial intelligence (AI) and precision medicine. The integration of vast amounts of genomic, transcriptomic, proteomic, metabolomic, imaging, and electronic health record (EHR) data will be used to enhance early diagnosis and to make more precise, personalized risk predictions based on AI and machine learning algorithms. Precision nephrology will allow the development of preventive and therapeutic strategies based on the individual's molecular characteristics, and not just on traditional clinical indicators. Moreover, liquid biopsy using circulating cell-free nucleic acids, EVs and urinary exosomes is a promising alternative, which is non-invasive and allows continuous disease monitoring. The use of integrated multi-biomarker panels (comprising molecular, clinical and imaging biomarkers) is likely to improve the accuracy of diagnosis and the ability to predict disease outcomes over single marker approaches. Additional studies should focus on large multicenter validations, harmonization of analytical methods, cost-effectiveness studies and regulatory harmonization to ensure potential routine clinical use. These developments revolutionize the care of DKD from reactive treatment to personalized, predictive, and proactive care, leading to better renal and cardiovascular outcomes in the long run.

8. Conclusion

Diabetic kidney disease (DKD) is one of the most serious and most common causes of chronic kidney disease and

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end-stage kidney failure in the world and is a complication of diabetes. Traditional markers of renal disease like albuminuria, estimated glomerular filtration rate (eGFR) and serum creatinine still have a major role in the clinical management of renal disease, but their limited sensitivity to detect early renal injury calls for an improved diagnostic strategy. Early work in the field of biomarkers has led to the identification of many potential markers of glomerular injury, tubular dysfunction, inflammation, oxidative stress, fibrosis and metabolic changes. In addition, advances in the fields of genomics, transcriptomics, proteomics, metabolomics, and multi-omics technologies have significantly increased the knowledge of DKD pathogenesis and allowed for the identification of highly sensitive molecular markers for early diagnosis and prognosis. The incorporation of artificial intelligence, machine learning and precision nephrology further enhances the promise of personalized risk predictions and biomarker-based therapeutic decision-making. However, there are still many obstacles to marker validation, standardization of assays, cost-effectiveness, regulatory approval, and marker use in routine clinical practice. These biomarkers must be introduced into clinical practice on a large scale and with standardized protocols before they can be broadly applied. In summary, the use of conventional clinical parameters and novel molecular markers holds promise to advance the early detection, more accurate prognosis, individualized therapeutic approach, and long-term renal and cardiovascular outcomes in diabetic kidney disease.

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