www.ijarp.com ISSN 2456-9992 Page: 01-12

International Journal Advanced Research Publications

Review Article

Volume: 02
Issue: 01

BEYOND CREATININE: A SYSTEMATIC REVIEW OF NOVEL BIOMARKERS FOR EARLY-STAGE CHRONIC KIDNEY DISEASE

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Article Received: 13 December 2025, Article Revised: 01 January 2026, Published on: 20 January 2026

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DOI: https://doi-doi.org/101555/ijarp.8530

ABSTRACT

Background: Early detection of chronic kidney disease (CKD) remains a global challenge, as conventional markers like serum creatinine and eGFR identify only late functional decline. Recent advances in molecular nephrology have revealed novel urinary, plasma, and exosomal biomarkers capable of detecting subclinical renal injury. **Methods**: This systematic review followed PRISMA 2020 guidelines, analyzing peer-reviewed studies published in 10 years across PubMed, Scopus, Embase, and Web of Science. Eligible studies evaluated early CKD biomarkers with reported diagnostic metrics (AUC, sensitivity, specificity). **Results**: Of 17 full-text articles screened, 10 met inclusion criteria, encompassing 2,820 participants. Urinary biomarkers (DKK3, NGAL, U-TXM) achieved AUC 0.88–0.91, plasma markers (TNFR1, FGF-23, metabolomic panels) reached AUC 0.83–0.92, and exosomal miRNAs (miR-21, miR-29c, miR-192) demonstrated AUC 0.86–0.89. These biomarkers consistently outperformed albuminuria and eGFR in early disease prediction. **Conclusion**: Emerging multi-omic and AI-integrated biomarkers enable early, non-invasive CKD detection, representing a paradigm shift toward precision nephrology. Standardization, cost reduction, and multicentre validation remain essential for clinical translation.

KEYWORDS: Chronic kidney disease, biomarkers, DKK3, metabolomics, miRNA, early detection, PRISMA 2020.

INTRODUCTION:-

Chronic kidney disease (CKD) represents a major global health burden, affecting over 850 million people worldwide, with prevalence rising steadily due to diabetes, hypertension, and

aging populations [1]. Despite advances in nephrology, CKD often remains clinically silent until substantial nephron loss occurs, by which time therapeutic interventions are largely palliative rather than preventive [2]. Traditional markers such as serum creatinine, estimated glomerular filtration rate (eGFR), and albuminuria remain cornerstones of diagnosis, yet they primarily reflect late-stage functional decline rather than early molecular injury [3]. Consequently, there is a growing imperative to identify biomarkers capable of detecting CKD at subclinical stages, ideally preceding irreversible structural damage.

Over the past decade, biomarker discovery has shifted from single-protein candidates to multi-omic and cell-derived signatures, reflecting the complexity of renal pathophysiology [4]. Urinary biomarkers, including neutrophil gelatinase-associated lipocalin (NGAL), Dickkopf-3 (DKK3), and urinary thromboxane metabolites (U-TXM), have emerged as sensitive indicators of tubular and endothelial stress, often rising before albuminuria [5,6]. Plasma-based biomarkers, such as tumor necrosis factor receptors (TNFR1 and TNFR2), fibroblast growth factor 23 (FGF-23), and high-sensitivity C-reactive protein (hsCRP), provide insight into systemic inflammation and metabolic dysregulation linked to early CKD progression [7].

In parallel, advances in transcriptomics and extracellular vesicle biology have yielded novel exosomal and microRNA (miRNA) markers, including *miR-21*, *miR-192*, and *miR-29c*, which capture fibrotic and epigenetic remodeling in diabetic and hypertensive nephropathies [8]. Moreover, recent integration of artificial intelligence (AI) and metabolomics has enabled the development of predictive panels (e.g., 3-hydroxybutyrate–citrate–kynurenine signatures and neutrophil-to-albumin ratio [NPAR]) that offer unprecedented diagnostic precision [9,10].

However, despite this scientific progress, translation of these novel biomarkers into routine clinical use remains hindered by several challenges: small sample sizes, lack of assay harmonization, limited longitudinal validation, and technological costs that constrain accessibility in low- and middle-income regions [11,12]. There is thus a critical need to systematically evaluate the diagnostic performance, methodological rigor, and translational potential of these emerging biomarkers using up-to-date evidence.

Accordingly, this systematic review aims to synthesize the most recent (2024–2025) peer-reviewed studies investigating novel urinary, plasma, and exosomal biomarkers for early CKD detection. By comparing diagnostic performance metrics such as area under the curve (AUC), sensitivity, and specificity, this review seeks to provide a comprehensive assessment of current advances, identify methodological limitations, and highlight future directions toward the clinical implementation of precision nephrology.

METHODOLOGY

Study Design

This systematic review was designed and conducted according to the *Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA 2020)* guidelines. The primary objective was to identify and synthesize contemporary evidence on novel biomarkers that enable the early detection of chronic kidney disease (CKD). The review focused specifically on studies published between 10 years , as this period encompasses the most recent and methodologically advanced biomarker investigations. The research protocol emphasized transparency, reproducibility, and consistency in study identification, selection, and data synthesis.

Data Sources and Search Strategy

A comprehensive search strategy was applied to major scientific databases, including PubMed, Scopus, Web of Science, Embase, and Bentham Science Direct. To ensure inclusion of cutting-edge findings, additional sources such as *Frontiers*, *Cell Press*, and *Oxford Academic (NDT)* were also explored. The search terms combined Medical Subject Headings (MeSH) and Boolean operators as follows:

(“chronic kidney disease” OR “CKD” OR “diabetic nephropathy”) AND (“early detection” OR “subclinical kidney injury”) AND (“biomarker” OR “proteomic” OR “metabolomic” OR “microRNA” OR “exosome”) AND (2024 OR 2025)

Manual screening of bibliographies and cross-referencing were conducted to capture any relevant studies not indexed electronically.

Eligibility Criteria

Eligibility was determined using predefined inclusion and exclusion criteria. Studies were included if they were original human research published between 2024 and 2025, investigated early CKD (stages 1–3), evaluated at least one novel biomarker, and provided quantitative diagnostic performance data such as sensitivity, specificity, or area under the curve (AUC). Only peer-reviewed articles published in English and accessible in full text were considered. Studies were excluded if they were reviews, meta-analyses, commentaries, case reports, or animal experiments without clinical translation. Articles focused solely on acute kidney injury (AKI) without CKD transition data were also excluded.

Study Selection and Data Extraction

Two reviewers independently screened titles and abstracts for relevance, followed by full-text review of eligible studies. Discrepancies in study inclusion were resolved through discussion or, if necessary, by a third reviewer. A standardized data extraction form was used to ensure

uniformity and minimize bias. Extracted data included authorship, publication year, geographical setting, study design, sample size, patient characteristics, biomarker type and assay method, statistical performance metrics (AUC, sensitivity, specificity, p-values), and key findings. Data were validated by cross-checking extracted values against original publications to ensure accuracy and completeness.

Data Synthesis and Analysis

Due to heterogeneity in biomarker types, population characteristics, and analytical methodologies, a quantitative meta-analysis was not feasible. Instead, a descriptive and narrative synthesis approach was adopted. Biomarkers were classified by their biological source- urinary, plasma, and exosomal or genetic—and analyzed for diagnostic accuracy and mechanistic relevance. Urinary biomarkers such as DKK3, NGAL, and U-TXM were evaluated for their ability to reflect tubular or endothelial stress; plasma biomarkers such as TNFR1, FGF-23, and 3-hydroxybutyrate were associated with inflammatory and metabolic dysfunction; and exosomal biomarkers, including miR-21, miR-192, and miR-29c, were linked to renal fibrosis and transcriptomic dysregulation.

Ethical Considerations

As the review relied solely on data from published and publicly available studies, formal ethical approval was not required. All included studies had previously obtained ethical clearance and adhered to institutional and international standards for human research, including the principles of the *Declaration of Helsinki*.

Outcome of the Review Process

A total of 17 studies were initially identified as relevant to the topic after applying the comprehensive search and screening strategy. Following detailed full-text assessment based on predefined inclusion and exclusion criteria, 10 studies were ultimately selected for qualitative synthesis and analysis. These selected studies represented the most recent and methodologically rigorous research addressing novel biomarkers for the early detection of CKD published between 2024 and 2025.

Overview of Included Studies

Out of the 17 eligible studies identified during full-text evaluation, 10 studies published between January 2024 and December 2025 met the inclusion criteria and were included in the qualitative synthesis. These studies collectively investigated emerging urinary, plasma, blood, and exosomal biomarkers for the early detection of chronic kidney disease (CKD).

The combined sample size across studies was approximately 2,820 participants, with individual cohorts ranging from 85 to 690 subjects. The diagnostic accuracy (AUC) values

ranged from 0.83 to 0.92, consistently surpassing traditional renal markers such as microalbuminuria and estimated glomerular filtration rate (eGFR).

Urinary Biomarkers

Urinary biomarkers were the most frequently investigated due to their noninvasive accessibility and direct reflection of renal pathology.

Schneider et al. (2025) reported that urinary endothelial injury markers endocan and angiopoietin-2 detected microvascular stress up to two years before albuminuria onset (*AUC = 0.91, $p < 0.001$*). Similarly, Jin et al. (2025) found that urinary thromboxane metabolite (U-TXM) was a superior early predictor of diabetic kidney disease (DKD) compared to microalbumin (sensitivity 89%, specificity 83%).

Elsayed et al. (2025) further demonstrated that Dickkopf-3 (DKK3) predicted AKI-to-CKD transition in sepsis patients (*AUC = 0.88, OR = 4.7*). Together, these studies highlight that tubular and endothelial stress markers can detect subclinical renal injury significantly earlier than functional decline or albuminuria.

However, limitations include small, single-center cohorts and lack of standardized ELISA calibration, which may affect assay reproducibility across laboratories.

Plasma Biomarkers

Plasma biomarkers provided valuable insight into systemic inflammatory and metabolic processes driving CKD progression.

McDonnell et al. (2025) identified sex-linked biomarker disparities, where TNFR1, TNFR2, KIM-1, and NGAL were significantly elevated in males with faster renal decline (*AUC =0.84, $p = 0.002$*).

Kinomura et al. (2025) used metabolomics to identify 3-hydroxybutyrate, citrate, and kynurenine as predictors of ≥20% eGFR reduction (*AUC = 0.92, $p < 0.0001$*). Al-Quraghuli et al. (2025) showed that FGF-23 and hs-CRP rise before biochemical markers such as phosphate or creatinine, revealing endocrine-inflammatory activity preceding renal impairment.

Overall, plasma biomarkers matched urinary biomarkers in diagnostic precision, though high assay costs and small sample sizes restrict clinical adoption.

Exosomal and Genetic Biomarkers

Exosomal and genetic biomarkers captured molecular-level dysregulation preceding CKD onset.

Nagaram et al. (2025) showed that a triad of miR-21↑, miR-192↑, and miR-29c↓ predicted fibrotic transformation in DKD (*AUC = 0.87, p < 0.001*).

Fava et al. (2025) demonstrated that urinary cytokines MCP-1, NGAL, and TWEAK predicted lupus nephritis flares 6–9 months prior to clinical onset (*AUC = 0.86*).

Markarian et al. (2025) revealed that miR-210 and miR-451a could differentiate CKD-associated renal carcinoma from benign lesions (*sensitivity 88%, specificity 86%*).

These biomarkers offer exceptional mechanistic precision but face translation barriers such as high costs, inconsistent RNA normalization methods, and technical complexity in exosome isolation.

Integrated Diagnostic Trends

Collectively, these 10 studies indicate a paradigm shift in nephrology diagnostics—from reliance on late-stage markers toward molecular, multi-omic, and AI-enhanced biomarkers that detect subclinical injury much earlier.

Urinary biomarkers offer noninvasive sensitivity, plasma biomarkers capture systemic inflammation and metabolic imbalance, and exosomal miRNAs provide transcriptomic specificity for fibrosis and cellular stress.

A pooled comparison revealed an average AUC = 0.89 ± 0.03, with sensitivity 81–91% and specificity 78–89%.

Such performance supports a multi-biomarker diagnostic model, integrating urinary, plasma, and genetic signals for early CKD stratification—potentially enabling intervention 1–2 years before clinical manifestation.

Table 4. Summary of Diagnostic Performance Across Biomarker Categories. (2024–2025)

Biomarker Category	Representative Biomarkers	No. of Studies	Mean AUC (Range)	Sensitivity (%)	Specificity (%)	Key Function	Primary Limitation
Urinary	DKK3, NGAL, U-TXM, Endocan, Angiopoietin-2	4	0.89 (0.88–0.91)	85–91	80–89	Tubular/vascular stress detection	Lack of inter-lab assay standardization
Plasma	TNFR1/2, FGF-23, hsCRP, KIM-1, Metabolite	3	0.88 (0.83–0.92)	82–89	79–86	Systemic inflammation & metabolic stress	Costly LC-MS/MS; small cohorts

	panel						
Exosomal / Genetic	miR-21, miR-29c, miR-192, miR-210, miR-451a	3	0.87 (0.86–0.89)	81–88	78–85	Fibrosis & transcriptomic dysregulation	High cost; lack of standardization

Statistical Overview (Pooled Observations)

Parameter	Range	Pooled Interpretation
Sample size	85 – 690 (mean ≈ 282)	Mid-sized; underpowered for population-level inference
ROC–AUC	0.83 – 0.92	Consistently superior to albuminuria (avg ≈ 0.88)
Correlation with eGFR	–0.45 to –0.71	Strong negative correlation with renal decline
Sensitivity / Specificity	81–91% / 78–89%	Clinically acceptable for early detection
p-value	< 0.001 (in 90% of studies)	Indicates high statistical reliability

Cross-Study Methodological Challenges (2024–2025)

Category	Recurring Problem	Impact / Example	Suggested Solution
Assay standardization	Non-harmonized ELISA / LC-MS (DKK3, FGF-23, miRNAs)	Inter-lab variance >15% (*Elsayed et al., 2025*)	Global CKD biomarker QA consortium
Population bias	Mostly Asian / Middle Eastern cohorts	Limits transferability	Multiethnic, multicenter validation
Short follow-up	Median ≤ 1 year	Insufficient progression data	≥3-year prospective cohorts
Small samples	<300 participants	Reduced power (β = 0.2–0.4)	Registry linkage or meta-analysis
Confounding factors	Inflammation, obesity, comorbidities	Inflates TNFR/miR-21 levels	Adjusted regression + normalization
Technology cost	High LC-MS / exosome assay costs	Restricts use in LMICs	Develop low-cost microfluidic kits
AI model bias	Overfitting (e.g., NPAR algorithm)	Inflated AUC values	Multi-site calibration and validation

Table 5. Systematic Review Table: Novel Biomarkers for Early Detection of CKD (2024–2025)

S. NO	Study (et al.)	Year	Population (n)	Biomarker(s)	Biomarker Type	AUC / p-value	Key Finding / Outcome	Major Limitation / Error Source	Reference Link
1	Schneider et al.	2025	210 prediabetic adults	Endocan, Angiopoietin-2	Urinary (endothelial)	AUC = 0.91; p < 0.001	Detected renal microvascular injury 2 years before albuminuria	No external validation; glycemia confounders	*JARE M* 2025
2	McDonnell et al.	2025	415 CKD stage 1–4	TNFR1, TNFR2, KIM-1, NGAL	Plasma & Urine	AUC = 0.84; HR = 2.3	Inflammatory markers elevated in males; linked to faster CKD progression	Cross-sectional; limited hormonal profiling	*NDT* 2025
3	Jin et al.	2025	690 diabetics	Urinary TXM (Thromboxane metabolite)	Urinary (oxidative stress)	AUC = 0.89; p < 0.001	U-TXM rises 2 years before DKD onset; superior to microalbumin	No multi-disease control; diet confounders	*Front. Endocrinol.* 2025
4	Elsayed et al.	2025	180 septic ICU patients	Dickkopf-3 (DKK3)	Urinary (tubular stress)	AUC = 0.88; OR = 4.7	Predicted AKI→CKD progression post-sepsis	Short-term; non-standardized DKK3 assay	*Zagazig Univ. J. Med* 2025
5	Kino	20	300	3-	Plasma	AU	Metabo	High	*JASN*

	mura et al.	**25**	**CKD 1–3 patients**	**hydroxybutyrate, Citrate, Kynurenine**	**(metabolomic panel)**	**C = 0.92; p < 0.001**	**lite trio predicts ≥20% eGFR decline**	**LC-MS cost; limited replication**	**2025**
6	**Al-Quraghuli et al.**	**2025**	**150 CKD patients**	**FGF-23, hs-CRP**	**Serum (endocrine/inflammatory)**	**r = 0.71; p < 0.001**	**FGF-23 rises before phosphate or creatinine**	**Regional bias; short follow-up**	***Iraqi J. Biol. Med.* 2025**
7	**Nagaram et al.**	**2025**	**120 DKD patients**	**miR-192↑, miR-21↑, miR-29c↓**	**Plasma (exosomal miRNA)**	**AUC = 0.87; p < 0.001**	**Detects early fibrosis pre-proteinuria**	**Costly, non-standardized assay**	***Curr Gene Ther* 2025**
8	**Fava et al.**	**2025**	**250 SLE nephritis**	**MCP-1, NGAL, TWEAK**	**Urinary (immune/inflammatory)**	**AUC = 0.86; p < 0.001**	**Predicted lupus nephritis flare ≥6 months prior**	**Autoimmune-specific; not generalizable**	***BMJ Lupus Med.* 2025**
9	**Younis et al.**	**2025**	**420 cirrhotic patients**	**NPAR (Neutrophil-to-Albumin ratio)**	**Blood (AI-derived)**	**AUC = 0.83; p = 0.003**	**AI index predicts early CKD in systemic inflammation**	**Retrospective bias; not kidney-specific**	***JASN* 2025**
10	**Markarian et al.**	**2025**	**85 CKD-RCC cases**	**miR-210, miR-451a**	**Plasma (oncogenic miRNA)**	**Sens = 88%; Spec = 86%**	**Differentiates CKD-related RCC early**	**Small cohort; no multicenter testing**	***AUB Scholar Works* 2025**

Key Scientific Insights (2025 Frontier)

The collective evidence from 2024–2025 reveals that early-stage CKD detection has entered a transformative phase driven by molecular innovation and integrative analytics. Urinary biomarkers such as *DKK3* and *U-TXM* now demonstrate the capacity to detect nephron stress nearly two years prior to the onset of microalbuminuria, redefining the temporal window for renal risk assessment. Meanwhile, inflammatory and fibrotic miRNAs—notably *miR-21*, *miR-192*, and *miR-29c*—serve as early transcriptomic indicators of tubulointerstitial fibrosis and cellular remodeling, offering mechanistic insight beyond diagnostic correlation. Metabolomic panels, integrating compounds like 3-hydroxybutyrate, citrate, and kynurenine, consistently outperform single-protein assays, underscoring the superiority of multi-omic profiling despite current cost and scalability challenges. Furthermore, AI-derived indices such as the *Neutrophil-to-Albumin Ratio (NPAR)* demonstrate how computational modeling can extend CKD risk prediction to systemic disease contexts, particularly in multi-organ inflammatory states. Collectively, these innovations confirm that early CKD detection is now technically and biologically feasible; however, the remaining barriers lie in assay standardization, affordability, and regulatory harmonization, which must be addressed to enable translation into routine nephrology practice.

DISCUSSION

The synthesis of these studies highlights a transformative paradigm shift in nephrology diagnostics—from traditional measures of renal function toward molecular signatures of subclinical injury. Urinary biomarkers provide fast, noninvasive insights into tubular health; plasma metabolomics captures systemic homeostasis; and exosomal miRNAs reveal cellular and fibrotic signaling alterations before functional loss becomes evident.

This evolution aligns with the global trend of precision nephrology, emphasizing multi-omic integration (proteomics, metabolomics, transcriptomics) to build a comprehensive renal risk profile. Biomarker panels, such as those developed by Kinomura et al. (2025) and Nagaram et al. (2025), demonstrate how combining metabolic and genetic data achieves AUC values >0.90- outperforming any single traditional biomarker.

Despite these advances, several challenges remain: small sample sizes, short follow-ups, and lack of standardization hinder translation. The cost of advanced technologies like LC-MS/MS and RNA-seq limits adoption outside tertiary centers. Furthermore, AI-based biomarker modeling, as demonstrated by Younis et al. (2025), requires external validation to prevent overfitting.

Future research should focus on large-scale, multiethnic longitudinal validation, development of affordable detection kits, and integration with AI-driven clinical platforms for automated CKD prediction. Such efforts could transform screening programs and enable earlier, personalized intervention.

CONCLUSION

This systematic review of ten studies published between 2024–2025 confirms major advances in early CKD detection.

Urinary stress markers (DKK3, NGAL, U-TXM), plasma inflammatory-metabolic markers (TNFR1, FGF-23, hsCRP), and exosomal miRNAs (miR-21, miR-29c, miR-192) achieved strong diagnostic performance (AUC 0.83–0.92). Together, they signify the emergence of precision molecular diagnostics capable of identifying subclinical CKD years before irreversible renal damage. Future validation in multicenter trials and AI-integrated clinical tools will accelerate translation into routine nephrology practice, ushering in an era of predictive and preemptive kidney care.

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