ISSN: 2181-3337 | SCIENTISTS.UZ VOLUME 5 ISSUE 8 AUGUST 2026

EARLY DETECTION OF CHRONIC KIDNEY DISEASE IN CHILDREN: RISK FACTORS AND CONTEMPORARY DIAGNOSTIC STRATEGIES: A NARRATIVE REVIEW

Akhmedova Dilorom[1], **Pulatova Maftunaxon**[2]
Tashkent State Medical University, Tashkent, Republic of Uzbekistan
Family Polyclinic No. 67, Tashkent City, Republic of Uzbekistan[1,2]

DOI: 10.67620/sai.58d0006

ABSTRACT

Pediatric chronic kidney disease (CKD) may remain minimally symptomatic while structural or tubular injury progresses, making early recognition essential. To synthesize risk factors, limitations of conventional detection, and emerging strategies for earlier pediatric CKD identification and risk stratification. This narrative review critically reorganizes the 46 publications in the source manuscript (2010-2025), including cohort, population-based, biomarker, imaging, review, and prediction-model studies. Because the source did not document a reproducible search strategy, formal eligibility criteria, or risk-of-bias assessment, no systematic-review claim is made. CAKUT, prior acute kidney injury, low birth weight, obesity, hypertension, nephrotoxic exposure, and reduced nephron reserve define important risk contexts. Creatinine-based eGFR remains essential but is complemented by urine protein or albumin quantification, cystatin C, tubular biomarkers, ultrasound and Doppler assessment, elastography, quantitative MRI, genomic testing, metabolomics, artificial intelligence, and prognostic models. The most defensible early-detection strategy is longitudinal and risk-oriented, integrating clinical history, kidney function, urine findings, blood pressure, and structural imaging. Emerging biomarkers and digital tools are promising but require further pediatric standardization and validation.

ACCEPTED

07.09.26

Keywords: *chronic kidney disease; children; early diagnosis; biomarkers; estimated glomerular filtration rate; pediatric nephrology.*

CC BY

Introduction

Chronic kidney disease (CKD) in childhood comprises a heterogeneous group of persistent structural and functional kidney abnormalities whose clinical consequences can extend across the life course. Unlike adult CKD, which is frequently driven by diabetes and long-standing vascular disease, pediatric CKD more often arises from congenital, hereditary, and developmental disorders. Its importance is amplified by the fact that kidney dysfunction occurs during periods of somatic growth, neurodevelopment, and maturation of multiple organ systems. Even moderate loss of kidney function may be accompanied by impaired growth and nutritional status, disordered mineral and bone metabolism, hypertension, and cardiovascular morbidity (Baiko, 2020; Karimdzhanov et al., 2019; Panzarino et al., 2022; Warady et al., 2023).

The epidemiology of pediatric CKD remains less precisely characterized than that of adult disease. Severe CKD is uncommon, early stages may be clinically silent, diagnostic criteria and estimated

glomerular filtration rate (eGFR) equations vary by age and setting, and population-based screening is inconsistent. Consequently, registry data tend to overrepresent children with advanced disease or kidney replacement therapy and underrepresent subclinical or early-stage CKD (Amanullah et al., 2022; Baiko, 2020; Panzarino et al., 2022). Access to prenatal diagnosis, laboratory testing, pediatric nephrology services, and long-term follow-up also influences when CKD is recognized, particularly in low- and middle-income settings (Amanullah et al., 2022; Çetinkaya et al., 2020).

Congenital anomalies of the kidneys and urinary tract (CAKUT) are a leading cause of pediatric CKD, but the etiologic spectrum also includes hereditary nephropathies, chronic glomerular disease, obstructive uropathy, consequences of acute kidney injury (AKI), nephrotoxic exposure, and increasingly recognized metabolic stressors such as obesity and hypertension (Çetinkaya et al., 2020; Geylis et al., 2023; Panzarino et al., 2022; Sawyer et al., 2023). The early clinical problem is that preserved nephron compensation can maintain filtration within an apparently acceptable range after structural or tubular injury has begun. Serum creatinine may therefore rise after the pathological process is already established, especially in children with low muscle mass or abnormal growth (den Bakker et al., 2022; Skidmore et al., 2024).

This review examines the major risk contexts for pediatric CKD, the limitations of conventional early detection, and the emerging role of biomarkers, advanced imaging, genomics, metabolomics, artificial intelligence, and clinical prediction tools. The central premise is that early diagnosis is most likely to improve when clinicians integrate longitudinal risk information rather than waiting for an isolated abnormal filtration marker.

Methods

Review Design and Evidence Base

This article is a narrative review developed from the evidence base contained in the source manuscript. Forty-six cited publications, published between 2010 and 2025, were organized into thematic domains: epidemiology and etiology; clinical risk factors for CKD development or progression; conventional laboratory assessment; emerging biomarkers; imaging; and genomic, metabolomic, artificial-intelligence, and prognostic approaches. The source literature includes narrative reviews, cohort and population-based studies, biomarker studies, imaging studies, and prediction-model research.

The source manuscript did not report a reproducible bibliographic search strategy, search dates, database coverage, formal inclusion or exclusion criteria, duplicate screening, risk-of-bias assessment, or evidence-grading framework. Accordingly, the present paper is explicitly framed as a narrative review rather than a systematic review or meta-analysis. No pooled effect estimates were calculated. Findings were synthesized qualitatively according to clinical relevance for early detection and risk stratification.

Results

Epidemiology, Etiology, and High-Risk Clinical Contexts

The available evidence supports a risk-first approach to case finding. CAKUT, including renal hypoplasia or dysplasia, obstructive uropathy, posterior urethral valves, multicystic dysplastic kidney, and a solitary functioning kidney, may initially coexist with apparently adequate filtration because of compensatory hyperfiltration. Reduced nephron mass, however, creates a biological substrate for later proteinuria, hypertension, and progressive nephrosclerosis (Çetinkaya et al., 2020; Groen In 't Woud et al., 2023; Viteri et al., 2021; Weaver et al., 2023; Yavuz et al., 2024).

AKI is another important pathway to chronic dysfunction. Pediatric data indicate that kidney injury may not be fully reversible even when serum creatinine returns toward baseline. Recurrent AKI, persistent acute kidney disease after AKI, and nephrotoxic medication exposure are associated with subsequent CKD risk or faster loss of kidney function (Ameta et al., 2023; Melhem et al., 2021; Patel et al., 2023; Sigurjonsdottir et al., 2018). Children with pre-existing CKD are particularly vulnerable to accelerated decline after additional AKI episodes (Melhem et al., 2021).

ISSN: 2181-3337 | SCIENTISTS.UZ

Perinatal and metabolic factors also contribute to risk. Low birth weight is associated with reduced kidney function and hypertension during adolescence, consistent with the concept of limited nephron reserve (Brathwaite et al., 2023). Obesity may promote glomerular hyperfiltration, metabolic and inflammatory stress, and albuminuria or proteinuria; it also complicates interpretation of kidney-function estimates because body composition can influence creatinine-based assessment (Barlabà et al., 2024; Carullo et al., 2023; Sawyer et al., 2023; van Dam et al., 2024). Hypertension is both a consequence of kidney disease and a mechanism of further injury. Ambulatory blood-pressure abnormalities, including nocturnal hypertension, may identify clinically important cardiovascular risk that is not captured by routine daytime measurements (Düzova et al., 2019; Karimdzhanov et al., 2019, 2024; Savenkova, 2017).

Table 1

Risk-oriented framework for early detection of pediatric chronic kidney disease

Risk context	Rationale for surveillance	Priority assessments	Representative evidence
CAKUT / reduced nephron mass	Structural abnormalities may precede functional decline; compensatory hyperfiltration can mask early loss of nephron reserve.	Longitudinal eGFR, urine protein/albumin, blood pressure, renal ultrasound; phenotype-specific follow-up.	Çetinkaya et al., 2020; Groen In 't Woud et al., 2023; Viteri et al., 2021
Prior AKI / nephrotoxic exposure	Incomplete recovery and recurrent injury can accelerate CKD development or progression.	Repeat kidney function after AKI, urine testing, blood pressure, medication review.	Ameta et al., 2023; Melhem et al., 2021; Patel et al., 2023
Low birth weight	Lower nephron endowment may reduce renal reserve and increase later hypertension risk.	Blood pressure, eGFR, urinalysis when additional risk factors are present.	Brathwaite et al., 2023
Obesity	Hyperfiltration and metabolic-inflammatory stress may produce albuminuria and complicate eGFR interpretation.	Blood pressure, urine albumin/protein, kidney-function assessment with attention to body composition.	Barlabà et al., 2024; Carullo et al., 2023; van Dam et al., 2024
Hypertension	May reflect kidney injury and independently accelerate progression; nocturnal abnormalities can be missed in clinic.	Repeated standardized blood pressure; ambulatory monitoring when indicated.	Düzova et al., 2019; Karimdzhanov et al., 2024
Persistent urinary abnormalities	Proteinuria/albuminuria can be an early marker and a progression factor; hematuria may indicate glomerular or genetic disease.	Confirm persistence; quantify protein/creatinine or albumin/creatinine; evaluate eGFR and blood pressure.	Kajiwara et al., 2020; Shanks et al., 2023

Note. The framework summarizes themes in the reviewed literature and is not a substitute for disease-specific pediatric nephrology guidelines.

Conventional Laboratory Assessment and Its Limitations

Routine urinalysis remains one of the most accessible routes to early detection. Proteinuria is both a marker of existing glomerular injury and a recognized risk factor for CKD progression. Quantification using a spot urine protein-to-creatinine ratio or albumin-to-creatinine ratio avoids the practical limitations of 24-hour urine collection and can support longitudinal monitoring (Baiko, 2020; Kajiwara et al., 2020; Vyalkova et al., 2013, 2019). School urine screening with quantitative protein-to-creatinine measurement has demonstrated the ability to identify a small number of children with clinically important, otherwise asymptomatic kidney disease (Kajiwara et al., 2020).

Proteinuria in children must be interpreted carefully because transient increases can occur during fever, exercise, or other physiologic stress. Persistent proteinuria, especially when accompanied by hematuria, hypertension, reduced eGFR, or structural kidney abnormalities, warrants more intensive evaluation. Conversely, CAKUT and tubulointerstitial disorders may produce important structural or

ISSN: 2181-3337 | SCIENTISTS.UZ

tubular injury before substantial albuminuria becomes evident (Chesnokova & Vyalkova, 2021; McLeod et al., 2020; Musiał & Zwolińska, 2018).

Serum creatinine and creatinine-based eGFR remain central to CKD staging and follow-up, but creatinine is affected by age, sex, muscle mass, growth, and nutritional status. A value within a population reference range does not necessarily exclude early kidney injury in an individual child (Baiko, 2020; den Bakker et al., 2022; Komarova et al., 2010; Skidmore et al., 2024). Cystatin C is less dependent on muscle mass and may improve assessment when creatinine is an unreliable surrogate of filtration, including in children with low muscle mass or atypical body composition. The evidence supports cystatin C as a complementary rather than isolated measure (den Bakker et al., 2022; Komarova et al., 2010; Skidmore et al., 2024).

Emerging Biomarkers and Molecular Phenotyping

An important direction in pediatric nephrology is the identification of biomarkers that reflect active nephron injury before marked filtration loss occurs. Urinary markers of tubular health, tubular injury, and inflammation have been associated with CKD progression in children. In the CKiD cohort, biomarker patterns involving epidermal growth factor, alpha-1 microglobulin, kidney injury molecule-1, monocyte chemoattractant protein-1, and YKL-40 provided information beyond conventional clinical variables, supporting the concept that urine can capture biological processes not fully represented by serum creatinine or eGFR (Greenberg et al., 2021).

Tubular biomarkers may be particularly relevant in obstructive uropathy, where longitudinal trajectories can reflect ongoing injury despite relatively stable conventional kidney-function measures (McLeod et al., 2020). Other candidate approaches include fractional excretion measures as indicators of tubular dysfunction, markers of inflammation and fibrogenesis, and growth differentiation factor 15 (Chesnokova et al., 2025; Chesnokova & Vyalkova, 2021; Musiał & Zwolińska, 2018; Thorsteinsdottir et al., 2020). These tools are biologically attractive but are not yet replacements for established CKD criteria because pediatric reference intervals, assay standardization, reproducibility, and incremental prognostic value require further validation.

Metabolomics extends this approach from individual biomarkers to molecular signatures. Machine-learning analysis of metabolomic profiles has distinguished etiologic patterns within pediatric CKD, while D-asparagine and the D/L-serine ratio have shown associations with chronic impairment of kidney function that may be less dependent on physique than conventional markers (Lee et al., 2022; Morishita et al., 2024). Such findings support molecular phenotyping as a future adjunct to clinical classification rather than a current population-screening strategy.

Imaging for Structural and Functional Characterization

Ultrasound remains the foundational imaging modality because it is widely available, nonionizing, and suitable for serial pediatric assessment. Clinically relevant features include kidney size relative to age and height, parenchymal thickness and echogenicity, corticomedullary differentiation, cystic change, collecting-system dilatation, and congenital structural abnormalities. Quantitative measures such as renal parenchymal area may contribute additional prognostic information in infants with CAKUT (Çetinkaya et al., 2020; Viteri et al., 2021; Yavuz et al., 2024).

Doppler ultrasound can extend conventional sonography by assessing intrarenal vascular parameters that may correlate with renal function and histopathological injury, although these measures should be interpreted in conjunction with clinical and laboratory findings (Sawires et al., 2018). Shear-wave elastography offers a noninvasive estimate of tissue stiffness and may capture remodeling or fibrosis. Pediatric studies have demonstrated diagnostic potential, but age-specific normative ranges and protocol standardization remain important limitations (Liu & Wang, 2021; Tiwari et al., 2025).

Quantitative magnetic resonance imaging (MRI) provides multiparametric information on renal tissue without ionizing radiation. Differences between healthy children and young people with CKD have

ISSN: 2181-3337 | SCIENTISTS.UZ

been demonstrated using quantitative MRI techniques, suggesting a role in noninvasive phenotyping and evaluation of selected patients with inconclusive conventional assessment (Dillman et al., 2022). Cost, scan duration, access, and the need for specialized expertise currently limit MRI as a broad screening modality.

Genomics, Artificial Intelligence, and Prognostic Modeling

Genomic sequencing can identify inherited causes of pediatric kidney disease that may not be apparent from routine clinical evaluation. In children referred for microscopic hematuria, genomic assessment has shown clinically meaningful diagnostic yield, illustrating the potential value of phenotype-directed genetic testing where hereditary disease is suspected (Shanks et al., 2023).

Artificial intelligence and statistical learning are also being applied to standard clinical data and imaging. Deep-learning features extracted from kidney ultrasound images in children with posterior urethral valves have been used to predict CKD progression, and combined clinical-imaging models may outperform either modality alone (Weaver et al., 2023). Large electronic health-record cohorts have also been used to characterize trajectories of kidney-function decline and identify clinical progression factors such as lower baseline eGFR, proteinuria, hypertension, disease etiology, and medical complexity (Gluck et al., 2023).

Prediction tools developed from pediatric CKD cohorts can integrate age, diagnosis, laboratory measurements, and longitudinal change to estimate time to kidney replacement therapy. These models illustrate a broader transition from threshold-based diagnosis toward dynamic risk estimation (Ng et al., 2023). Their clinical value will depend on external validation, transportability across health systems and populations, calibration over time, and transparent integration into clinical decision making.

Discussion

Toward an Integrated Risk-Oriented Diagnostic Strategy

The reviewed evidence supports a conceptual shift in pediatric CKD detection. A model centered primarily on elevated serum creatinine or an advanced reduction in eGFR is inherently late for a disease in which structural, tubular, vascular, or molecular abnormalities may precede clear filtration failure. A more defensible strategy begins by identifying children with increased pretest probability of kidney injury and then applying longitudinal multimodal surveillance (Baiko, 2020; den Bakker et al., 2022; Warady et al., 2023).

At the clinical level, this means prioritizing children with CAKUT, a solitary functioning kidney, prior AKI, recurrent nephrotoxic exposure, low birth weight, obesity, hypertension, persistent urinary abnormalities, or suspected hereditary disease. Baseline and follow-up evaluation should integrate eGFR, quantitative proteinuria or albuminuria, blood pressure, and structural imaging rather than interpreting any of these measurements in isolation. The association of proteinuria and hypertension with more rapid CKD progression in real-world pediatric cohorts further supports attention to these markers as both diagnostic and prognostic variables (Gluck et al., 2023; Groen In 't Woud et al., 2023).

Advanced tests should be selected according to the clinical question. Cystatin C is most useful when creatinine-based filtration assessment is uncertain; tubular biomarkers may help characterize active injury; elastography and quantitative MRI may refine structural or fibrotic phenotyping; and genomic sequencing may be most informative when the phenotype suggests inherited disease. Metabolomic and artificial-intelligence tools remain promising for future risk stratification but should not yet be treated as universal screening tests (Lee et al., 2022; Shanks et al., 2023; Tiwari et al., 2025; Weaver et al., 2023).

Clinical Implications

Three practical implications emerge. First, early detection should be longitudinal: repeated measurements are more informative than isolated normal values because pediatric CKD evolves over time and compensatory physiology may initially obscure loss of nephron reserve. Second, surveillance intensity should be proportional to risk. Children with structural anomalies, prior AKI, persistent proteinuria,

ISSN: 2181-3337 | SCIENTISTS.UZ

hypertension, or multiple interacting risk factors warrant closer follow-up than children without these features. Third, emerging technologies should be used to answer specific diagnostic or prognostic uncertainties rather than added indiscriminately. This tiered approach may improve detection while limiting unnecessary testing and cost.

Limitations of the Evidence Base

This review has several limitations. Most importantly, it is a narrative synthesis of the literature contained in the source manuscript and not a systematic review. The absence of a documented comprehensive search strategy means that relevant studies may be missing and publication-selection bias cannot be excluded. The cited evidence is heterogeneous with respect to CKD etiology, age, disease severity, outcome definitions, biomarker assays, imaging protocols, and follow-up duration. Many emerging biomarkers and imaging methods have been evaluated in relatively small or selected pediatric cohorts, limiting generalizability. Finally, technological performance does not automatically establish clinical utility; future studies must show that new tests improve decisions and patient outcomes beyond established risk factors and conventional measurements.

Future Research

Future pediatric CKD research should prioritize prospective multicenter validation of integrated risk models; age- and development-specific reference ranges for cystatin C and emerging biomarkers; standardized protocols for elastography and quantitative MRI; evaluation of genomic and metabolomic testing in clearly defined clinical pathways; and external validation of artificial-intelligence models across diverse populations and health systems. Research should also address implementation in low-resource settings, where simple risk stratification, blood-pressure measurement, urine testing, and accessible ultrasound may have greater immediate population impact than high-cost technologies (Amanullah et al., 2022).

Conclusion

Pediatric CKD can remain clinically silent while structural and molecular kidney injury progresses. The most clinically useful early-detection strategy is therefore not a single novel test but an integrated, risk-oriented model that combines medical history, eGFR, proteinuria or albuminuria, blood pressure, and renal imaging over time. Cystatin C, tubular biomarkers, advanced imaging, genomics, metabolomics, artificial intelligence, and prognostic models can add value in selected contexts, but most require additional pediatric standardization and validation. Earlier identification of kidney injury creates an opportunity for timely nephroprotective management before substantial irreversible nephron loss occurs.

Declarations

Ethics statement: This narrative review does not report new research involving human participants or animals.

Funding: Funding information was not provided in the source manuscript and should be confirmed before journal submission.

Conflict of interest: Author conflict-of-interest declarations were not provided in the source manuscript and should be completed before journal submission.

Author contributions: A formal contributor-role statement was not provided in the source manuscript and should be completed before journal submission.

REFERENCES

1. Amanullah, F., Malik, A. A., & Zaidi, Z. (2022). Chronic kidney disease causes and outcomes in children: Perspective from a LMIC setting. *PLOS ONE*, *17*(6), e0269632.
2. Ameta, P., Stoops, C., & Askenazi, D. J. (2023). Risk of chronic kidney disease in children who developed acute kidney injury secondary to nephrotoxic medication exposure in infancy. *Renal Failure*, *45*(1), 2218486.

ISSN: 2181-3337 | SCIENTISTS.UZ

3. Baiko, S. V. (2020). Chronic kidney disease in children: Definition, classification, and diagnostics. *Nephrology and Dialysis*, *22*(1), 53-70. https://doi.org/10.28996/2618-9801-2020-1-53-70
4. Barlabà, A., Grella, C., Tammaro, M., Petrone, D., Guarino, S., Miraglia Del Giudice, E., Marzuillo, P., & Di Sessa, A. (2024). Kidney function evaluation in children and adolescents with obesity: A not-negligible need. *European Journal of Pediatrics*, *183*(9), 3655-3664. https://doi.org/10.1007/s00431-024-05641-0
5. Boynton, S. A., Matheson, M. B., Ng, D. K., Hidalgo, G., Warady, B. A., Furth, S. L., & Atkinson, M. A. (2022). The relationship between neighborhood disadvantage and kidney disease progression in the Chronic Kidney Disease in Children (CKiD) cohort. *American Journal of Kidney Diseases*, *80*(2), 207-214. https://doi.org/10.1053/j.ajkd.2021.12.008
6. Brathwaite, K. E., Levy, R. V., Sarathy, H., Agalliu, I., Johns, T. S., Reidy, K. J., Fadrowski, J. J., Schwartz, G. J., Kaskel, F. J., & Melamed, M. L. (2023). Reduced kidney function and hypertension in adolescents with low birth weight, NHANES 1999-2016. *Pediatric Nephrology*, *38*(9), 3071-3082. https://doi.org/10.1007/s00467-023-05958-2
7. Carullo, N., Zicarelli, M., Michael, A., Faga, T., Battaglia, Y., Pisani, A., Perticone, M., Costa, D., Ielapi, N., Coppolino, G., Bolignano, D., Serra, R., & Andreucci, M. (2023). Childhood obesity: Insight into kidney involvement. *International Journal of Molecular Sciences*, *24*(24), 17400. https://doi.org/10.3390/ijms242417400
8. Çetinkaya, P. G., Gülhan, B., Düzova, A., Beşbaş, N., Hayran, M., Topaloğlu, R., & Özaltın, F. (2020). Clinical characteristics of children with congenital anomalies of the kidney and urinary tract and predictive factors of chronic kidney disease. *Turkish Journal of Pediatrics*, *62*(5), 746-755. https://doi.org/10.24953/turkjped.2020.05.005
9. Chesnokova, S. A., & Vyalkova, A. A. (2021). Pathogenetic markers for the diagnosis and prediction of chronic kidney disease in children. *Russian Bulletin of Perinatology and Pediatrics*, *66*(3), 62-69. https://doi.org/10.21508/1027-4065-2021-66-3-62-69
10. Chesnokova, S. A., Atyakshin, D. A., Vyalkova, A. A., & Davydova, N. O. (2025). Chronic kidney disease in children: Pathophysiological mechanisms and new approaches to the diagnosis of tubulointerstitial fibrosis. *Russian Bulletin of Perinatology and Pediatrics*, *70*(2), 19-29. https://doi.org/10.21508/1027-4065-2025-70-2-19-29
11. den Bakker, E., Bökenkamp, A., & Haffner, D. (2022). Assessment of kidney function in children. *Pediatric Clinics of North America*, *69*(6), 1017-1035.
12. Dillman, J. R., Benoit, S. W., Gandhi, D. B., Trout, A. T., Tkach, J. A., VandenHeuvel, K., & Devarajan, P. (2022). Multiparametric quantitative renal MRI in children and young adults: Comparison between healthy individuals and patients with chronic kidney disease. *Abdominal Radiology*, *47*(5), 1840-1852. https://doi.org/10.1007/s00261-022-03456-x
13. Düzova, A., Karabay Bayazit, A., Canpolat, N., Niemirska, A., Kaplan Bulut, I., Azukaitis, K., Karagoz, T., Oguz, B., Erdem, S., Anarat, A., Ranchin, B., Shroff, R., Djukic, M., Harambat, J., Yilmaz, A., Yildiz, N., Ozcakar, B., Büscher, A., Lugani, F., . . . Wühl, E. (2019). Isolated nocturnal and isolated daytime hypertension associate with altered cardiovascular morphology and function in children with chronic kidney disease: Findings from the Cardiovascular Comorbidity in Children with Chronic Kidney Disease study. *Journal of Hypertension*, *37*(11), 2247-2255. https://doi.org/10.1097/HJH.0000000000002160
14. Geylis, M., Coreanu, T., Novack, V., & Landau, D. (2023). Risk factors for childhood chronic kidney disease: A population-based study. *Pediatric Nephrology*, *38*(5), 1569-1576.
15. Gluck, C. A., Forrest, C. B., Davies, A. G., Maltenfort, M., Mcdonald, J. R., Mitsnefes, M., Dharnidharka, V. R., Dixon, B. P., Flynn, J. T., Somers, M. J., Smoyer, W. E., Neu, A., Hovinga, C. A., Skversky, A. L., Eissing, T., Kaiser, A., Breitenstein, S., Furth, S. L., & Denburg, M. R. (2023).

Evaluating kidney function decline in children with chronic kidney disease using a multi-institutional electronic health record database. *Clinical Journal of the American Society of Nephrology*, *18*(2), 173-182. https://doi.org/10.2215/CJN.0000000000000051

16. Greenberg, J. H., Abraham, A. G., Xu, Y., Schelling, J. R., Feldman, H. I., Sabbisetti, V. S., Ix, J. H., Jogalekar, M. P., Coca, S., Waikar, S. S., Shlipak, M. G., Warady, B. A., Vasan, R. S., Kimmel, P. L., Bonventre, J. V., Denburg, M., Parikh, C. R., & Furth, S. (2021). Urine biomarkers of kidney tubule health, injury, and inflammation are associated with progression of CKD in children. *Journal of the American Society of Nephrology*, *32*(10), 2664-2677. https://doi.org/10.1681/ASN.2021010094
17. Groen In 't Woud, S., Roeleveld, N., Westland, R., Renkema, K. Y., Steffens, M. G., Gracchi, V., Lilien, M. R., van Wijk, J. A. E., Feitz, W. F. J., Schreuder, M. F., & van der Zanden, L. F. M. (2023). Uncovering risk factors for kidney injury in children with a solitary functioning kidney. *Kidney International*, *103*(1), 156-165. https://doi.org/10.1016/j.kint.2022.09.028
18. Kajiwara, N., Hayashi, K., Fujiwara, M., Nakayama, H., & Ozaki, Y. (2020). Identification of children with chronic kidney disease through school urinary screening using urinary protein/creatinine ratio measurement: An observational study. *Clinical and Experimental Nephrology*, *24*(5), 450-457. https://doi.org/10.1007/s10157-020-01852-5
19. Karimdzhanov, I. A., Iskanova, G. Kh., & Israilova, N. A. (2019). Chronic kidney disease in children: Problems of arterial hypertension. *Nephrology*, *23*(5), 47-55. https://doi.org/10.24884/1561-6274-2019-23-5-47-55
20. Karimdzhanov, I. A., Yusupova, G. A., Iskanova, G. Kh., & Israilova, N. A. (2024). Arterial hypertension as a risk factor for chronic kidney disease in children with congenital anomalies of the kidney and urinary tract (CAKUT) and its correction. *Nephrology*, *28*(1), 43-49. https://doi.org/10.36485/1561-6274-2024-28-1-43-49
21. Komarova, O. V., Tsygin, A. N., Kucherenko, A. G., & Smirnov, I. E. (2010). Cystatin C as a marker of kidney function in children with CKD. *Nephrology and Dialysis*, *12*(4), 271-274.
22. Lee, A. M., Hu, J., Xu, Y., Abraham, A. G., Xiao, R., Coresh, J., Rebholz, C., Chen, J., Rhee, E. P., Feldman, H. I., Ramachandran, V. S., Kimmel, P. L., Warady, B. A., Furth, S. L., & Denburg, M. R. (2022). Using machine learning to identify metabolomic signatures of pediatric chronic kidney disease etiology. *Journal of the American Society of Nephrology*, *33*(2), 375-386. https://doi.org/10.1681/ASN.2021040538
23. Liu, Q., & Wang, Z. (2021). Diagnostic value of real-time shear wave elastography in children with chronic kidney disease. *Clinical Hemorheology and Microcirculation*, *77*(3), 287-293.
24. McLeod, D. J., Sebastião, Y. V., Ching, C. B., Greenberg, J. H., Furth, S. L., & Becknell, B. (2020). Longitudinal kidney injury biomarker trajectories in children with obstructive uropathy. *Pediatric Nephrology*, *35*(10), 1907-1914. https://doi.org/10.1007/s00467-020-04602-7
25. Melhem, N., Rasmussen, P., Joyce, T., Clothier, J., Reid, C. J. D., Booth, C., & Sinha, M. D. (2021). Acute kidney injury in children with chronic kidney disease is associated with faster decline in kidney function. *Pediatric Nephrology*, *36*(5), 1279-1288. https://doi.org/10.1007/s00467-020-04777-z
26. Morishita, T., Nishizaki, N., Taniguchi, S., Sakai, S., Kimura, T., Mita, M., Nakagawa, M., Endo, A., Ohtomo, Y., Yasui, M., Shimizu, T., & Sasabe, J. (2024). Plasma D-asparagine and the D/L-serine ratio reflect chronic kidney diseases in children regardless of physique. *Amino Acids*, *56*(1), 38. https://doi.org/10.1007/s00726-024-03400-x
27. Musiał, K., & Zwolińska, D. (2018). Fractional excretion as a new marker of tubular damage in children with chronic kidney disease. *Clinica Chimica Acta*, *480*, 99-106. https://doi.org/10.1016/j.cca.2018.02.001
28. Ng, D. K., Matheson, M. B., Schwartz, G. J., Wang, F. M., Mendley, S. R., Furth, S. L., & Warady, B. A. (2023). Development of an adaptive clinical web-based prediction tool for kidney replacement

ISSN: 2181-3337 | SCIENTISTS.UZ

therapy in children with chronic kidney disease. *Kidney International*, *104*(5), 985-994. https://doi.org/10.1016/j.kint.2023.06.020

29. Panzarino, V., Lesser, J., & Cassani, F. A. (2022). Pediatric chronic kidney disease. *Advances in Pediatrics*, *69*(1), 123-132.
30. Patel, M., Hornik, C., Diamantidis, C., Selewski, D. T., & Gbadegesin, R. (2023). Patient level factors increase risk of acute kidney disease in hospitalized children with acute kidney injury. *Pediatric Nephrology*, *38*(10), 3465-3474. https://doi.org/10.1007/s00467-023-05997-9
31. Savenkova, N. D. (2017). Nephrogenic arterial hypertension in children and adolescents: Causes, classification, and diagnosis. *Russian Bulletin of Perinatology and Pediatrics*, *62*(4), 43-48. https://doi.org/10.21508/1027-4065-2017-62-4-43-48
32. Sawires, H., Salah, D., Hashem, R., Ismail, W., Salem, A., Botros, O., & Seif, H. (2018). Renal ultrasound and Doppler parameters as markers of renal function and histopathological damage in children with chronic kidney disease. *Nephrology*, *23*(12), 1116-1124. https://doi.org/10.1111/nep.13201
33. Sawyer, A., Zeitler, E., Trachtman, H., & Bjornstad, P. (2023). Kidney considerations in pediatric obesity. *Current Obesity Reports*, *12*(3), 332-344.
34. Shanks, J., Butler, G., Cheng, D., Jayasinghe, K., & Quinlan, C. (2023). Clinical and diagnostic utility of genomic sequencing for children referred to a Kidney Genomics Clinic with microscopic haematuria. *Pediatric Nephrology*, *38*(8), 2623-2630. https://doi.org/10.1007/s00467-022-05846-1
35. Sigurjonsdottir, V. K., Chaturvedi, S., Mammen, C., & Sutherland, S. M. (2018). Pediatric acute kidney injury and the subsequent risk for chronic kidney disease: Is there cause for alarm? *Pediatric Nephrology*, *33*(11), 2047-2055.
36. Skidmore, M., Spencer, S., Desborough, R., Kent, D., & Bhandari, S. (2024). Cystatin C as a marker of kidney function in children. *Biomolecules*, *14*(8), 938. https://doi.org/10.3390/biom14080938
37. Thorsteinsdottir, H., Salvador, C. L., Mjøen, G., Lie, A., Sugulle, M., Tøndel, C., Brun, A., Almaas, R., & Bjerre, A. (2020). Growth differentiation factor 15 in children with chronic kidney disease and after renal transplantation. *Disease Markers*, *2020*, 6162892. https://doi.org/10.1155/2020/6162892
38. Tiwari, K., Mittal, A., Sureka, B., Narayanan, D., Nalwa, A., Vishwajeet, V., Banerjee, M., Goel, A. D., Varthya, S. B., Tiwari, K., Sinha, A., & Singh, K. (2025). Utility of shear wave elastography in evaluation of children with chronic kidney disease. *Pediatric Nephrology*, *40*(6), 2021-2028. https://doi.org/10.1007/s00467-024-06573-5
39. Tolganbaeva, A. A., & Chingaeva, G. N. (2019). Chronic kidney disease in children: A modern view (literature review). *Bulletin of KazNMU*, *1*, 185-190.
40. van Dam, M. J. C. M., Pottel, H., Delanaye, P., & Vreugdenhil, A. C. E. (2024). The evaluation of kidney function estimation during lifestyle intervention in children with overweight and obesity. *Pediatric Nephrology*, *39*(11), 3271-3278.
41. Viteri, B., Elsingergy, M., Roem, J., Ng, D., Warady, B., Furth, S., & Tasian, G. (2021). Ultrasound-based renal parenchymal area and kidney function decline in infants with congenital anomalies of the kidney and urinary tract. *Seminars in Nephrology*, *41*(5), 427-433. https://doi.org/10.1016/j.semnephrol.2021.09.004
42. Vyalkova, A. A., Zorin, I. V., Chesnokova, S. A., & Plotnikova, S. V. (2019). Chronic kidney disease in children. *Nephrology*, *23*(5), 29-46. https://doi.org/10.24884/1561-6274-2019-23-5-29-46
43. Vyalkova, A. A., Zorin, I. V., Gordienko, L. M., Meshcheryakova, A. I., Egorochkina, M. S., & Savelyeva, E. V. (2013). Issues in the diagnosis of chronic kidney disease in children. *Practical Medicine*, *6*(75), 72-77.
44. Warady, B. A., Feldman, D. L., Bell, L. E., Bacchetta, J., Denburg, M. R., Flynn, J. T., Haffner, D., Johnson, R. J., Mitsnefes, M. M., Schaefer, F., Jaure, A., & Furth, S. L. (2023). Improving clinical

care for children with CKD: A report from a National Kidney Foundation scientific workshop. *American Journal of Kidney Diseases*, *81*(4), 466-474. https://doi.org/10.1053/j.ajkd.2022.09.017

45. Weaver, J. K., Milford, K., Rickard, M., Logan, J., Erdman, L., Viteri, B., D'Souza, N., Cucchiara, A., Skreta, M., Keefe, D., Shah, S., Selman, A., Fischer, K., Weiss, D. A., Long, C. J., Lorenzo, A., Fan, Y., & Tasian, G. E. (2023). Deep learning imaging features derived from kidney ultrasounds predict chronic kidney disease progression in children with posterior urethral valves. *Pediatric Nephrology*, *38*(3), 839-846. https://doi.org/10.1007/s00467-022-05677-0
46. Yavuz, S., Kıyak, A., & Sander, S. (2024). Outcome of children with multicystic dysplastic kidney: Does involved side matter? *Birth Defects Research*, *116*(1), e2297. https://doi.org/10.1002/bdr2.2297