Review Article

New Guideline for Chronic Kidney Disease 2024, What Primary Care Can Do About It?: A Narrative Review

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ABSTRACT

Background: Chronic Kidney Disease (CKD) presents an urgent global public health crisis, affecting over 850 million people worldwide, with low-income nations like Indonesia facing a high burden of undiagnosed cases due to limited awareness and a deficient primary care system.
Objective: This paper serves as a vital, practical response to the novelty of the KDIGO 2024 Clinical Practice Guideline update, which incorporates a decade of new evidence, including the ethical imperative to eliminate the ethnic coefficient from eGFR equations and the introduction of consensus-based "Practice Points."
Methods: This narrative review synthesizes the updated KDIGO 2024 Clinical Practice Guideline for the evaluation and management of CKD.
Results: Key findings from this review highlight that CKD diagnosis is not solely reliant on Glomerular Filtration Rate (GFR), but also on persistent markers of kidney damage such as albuminuria and urine sediment abnormalities. The 2024 updates strongly recommend the ethnicity-free CKD-EPI 2021 equation for routine screening, the use of estimated GFR based on creatinine and cystatin C (eGFRcr-cys) for superior accuracy, and the strong recommendation for Sodium-Glucose Cotransporter-2 Inhibitors (SGLT2i) in Type 2 Diabetes patients with CKD. Additionally, the guidelines introduce actionable risk prediction thresholds for nephrology referral, alongside practical advice like "sick day rules" for primary care.
Conclusions: It concludes that primary care, as the frontline in health services, must rapidly adopt these standards to enhance early screening, improve patient risk stratification, and facilitate timely, informed referrals to advanced care, thereby mitigating disease progression and improving patient outcomes globally.
Keywords: Chronic Kidney Disease, eGFR, KDIGO, Primary Care.

Introduction

The 2024 Kidney Disease Improving Global Outcomes (KDIGO) guideline updates optimizes services and applications of the latest science for Chronic Kidney Disease (CKD) patients.[1,2] Significant changes that 6 statements were left from the guideline in 2012 and updated both the approach to diagnosis, risk stratification to services for CKD patients.[1] Chronic Kidney Disease defined as abnormalities of kidney structure OR function, present for a minimum of 3 months, with implication for health.[1] Globally, in 2017, a systematic analysis found the prevalence of CKD to be 9.1% (8.5%-9.8%) with 697.5 million cases of CKD at all stages.[3] As of 2021, data from multiple international collegia of specialists revealed that there were over 850 million cases of

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kidney disease worldwide, nearly twice as many as patients with diabetes (422 million) and 20 times more than those with cancer (42 million) or HIV (36.7 million).[1] A multicenter study at a tertiary health service in Jakarta, Indonesia, from December 2021 to July 2022 found 1,152 patients with kidney failure.[4] This demonstrates that a rise in kidney disease cases, particularly CKD, is expected to occur for a number of reasons, including lifestyle choices and other factors.

Kidney illness is thought to affect 850 million people globally, with the majority residing in lower-middle-class and low-income nations (for example, Indonesia). In settings with limited resources and a deficient primary care system, up to 90% of people with CKD are not aware that they have the disease and do not seek treatment.[5] In a country like Indonesia, the role of primary care is needed for CKD cases, because the community, in addition to the lack of awareness of CKD and the lack of accessibility of primary care, is a cause of the increase in CKD cases in low-income countries. Therefore, Primary care, as the frontline in health care, needs to update its capacity and capability towards CKD through the guideline update of KDIGO 2024. This is because primary care will be the first place patients come before they can get referrals to advanced hospitals. Hence, it is important for primary care doctors to be familiar with the updates of KDIGO 2024 as well as the identification (screening) of CKD patients in primary care so that they can be referred more quickly to get complete health services.

CKD IN NOWADAYS: UPDATES FROM KDIGO 2024

The KDIGO 2024 Clinical Practice Guideline for the Evaluation and Management of CKD[1] significantly updates its 2012 predecessor,[2] incorporating a decade of new evidence to provide more precise guidance. A key methodological enhancement is the introduction of "Practice Points", which are consensus-based expert statements offering practical guidance for clinical questions lacking systematic reviews or to aid the implementation of graded recommendations. Unlike the "Not Graded" statements in KDIGO 2012,[2] Practice Points are now recognized as important expert guidance, not a lesser form of recommendation. Both guidelines maintain the CGA (Cause, GFR category, Albuminuria category) classification system for CKD, with KDIGO 2024[1] reinforcing its widespread acceptance and utility in guiding management and risk assessment. For assessing kidney function, while creatinine-based eGFR (eGFRcr) remains the initial assessment, KDIGO 2024 now strongly recommends (1B) the use of estimated GFR based on both creatinine and cystatin C (eGFRcr-cys) when cystatin C is available, citing its superior accuracy in diagnosis and staging of CKD and emphasizing understanding the implications of differences between eGFRcr and eGFRcys. Notably, the updated guideline explicitly advises against using ethnicity in eGFR computation.[1]

A major leap in KDIGO 2024 lies in risk prediction for kidney failure.[1] The guideline now strongly recommends (1A) employing externally validated risk equations to estimate the absolute risk of kidney failure in individuals with CKD G3–G5, establishing actionable thresholds for care. For instance, a 5-year kidney failure risk of 3–5% prompts nephrology referral, a 2-year risk >10% suggests multidisciplinary care, and a 2-year risk >40% indicates the need for kidney replacement therapy (KRT) modality education and transplant planning. Pharmacologically, Sodium-Glucose Cotransporter-2 Inhibitors (SGLT2i) are a significant addition, strongly recommended (1A) for adults with Type 2 Diabetes and CKD with an eGFR ≥20 mL/min/1.73 m^2, continuing even if eGFR falls lower.[1] Non-steroidal mineralocorticoid receptor antagonists (MRA) are also suggested for high-risk T2D patients with persistent albuminuria, with careful potassium monitoring. While KDIGO 2012 found insufficient evidence for uric acid-lowering to delay CKD progression, KDIGO 2024 now recommends (1C) uric acid-lowering for symptomatic hyperuricemia, but not for asymptomatic cases, to slow progression. Updates also include nuanced guidance on RAS inhibitor use, such as investigating >30% eGFR

decrease rather than immediately stopping for creatinine increases.[1]

The 2024 guideline introduces a dedicated chapter on medical management and drug stewardship, underscoring the necessity of periodic medication reviews, particularly during care transitions. A key practical update includes "sick day rules," advising planned temporary discontinuation of specific medications (e.g., SGLT2i, ACEi, ARBs, metformin) before elective surgery or during acute illness, coupled with clear communication on when to restart to mitigate harm. This promotes patient education on medication benefits and risks and encourages collaboration with pharmacists. Regarding optimal models of CKD care, the guideline reinforces the value of team-based, multidisciplinary care, providing specific risk-based criteria for initiating such comprehensive support. It further addresses modern care delivery, including the integration of telehealth technologies for patient education and remote monitoring, and offers detailed guidance on the transition of care for young people moving from pediatric to adult nephrology services. These updates collectively foster a more patient-centered, integrated, and evidence-based approach to CKD management, aiming to improve outcomes globally.[1]

DETECTION OF CKD, IS IT ONLY RELYING ON GFR?

According to KDIGO 2024, CKD is diagnosed when evidence of kidney damage or decreased kidney function persists for a minimum duration of three months. The diagnosis can be based on either one or more markers of kidney damage or a reduction in glomerular filtration rate (GFR). Markers of kidney damage include albuminuria (albumin-to-creatinine ratio [ACR] > 30 mg/g or > 3 mg/mmol), urine sediment abnormalities, persistent hematuria, electrolyte and other abnormalities attributable to tubular disorders, histological abnormalities, structural abnormalities identified by imaging, or a history of kidney transplantation. Alternatively, CKD can also be diagnosed when there is a decreased GFR of less than 60 ml/min/1.73 m^2 (corresponding to GFR categories G3a–G5), even in the absence of other markers of kidney damage, provided that this reduction is sustained for at least three months.[1]

Albuminuria is one of the critical markers in the detection, evaluation, and management of CKD. It refers to the presence of albumin in the urine. Under normal circumstances, the kidneys filter waste products while retaining essential proteins like albumin in the bloodstream. When the kidneys' filtering units (glomeruli) are damaged, they may allow albumin to leak into the urine, resulting in albuminuria.[1]

Building upon this understanding, numerous studies have consistently recognized albuminuria as a fundamental marker for diagnosing and managing CKD. According to the American Academy of Family Physicians (AAFP), persistently elevated serum creatinine and albuminuria serve as diagnostic and prognostic hallmarks of CKD, with even low levels of albuminuria being associated with adverse renal and cardiovascular outcomes.[6] Similarly, the Clinical Journal of the American Society of Nephrology highlights that glomerular filtration rate (GFR) and albuminuria are the primary measures for detecting, staging, and managing both acute and chronic kidney disease.[7] Furthermore, recent literature reinforces albuminuria as a strong indicator of kidney damage and a predictor of disease progression and cardiovascular complications.[8] Collectively, these findings emphasize its pivotal role in CKD assessment and care.

To standardize its application in clinical practice, albuminuria levels are categorized and interpreted to determine CKD stage and evaluate the risk of disease progression. According to KDIGO guidelines, albuminuria is classified into:

Table 1. Albuminuria Severity Grade[1]

Category	AER	ACR (approximately equivalent)		Terms
	(mg/24 h)	(mg/mmol)	(mg/g)	
A1	<30	<3	<30	Normal to mildly increased
A2	30-300	3-30	30-300	Moderately increased
A3	>300	>30	>300	Severely increased

Higher levels of albumin in urine have been strongly linked to an increased risk of CKD progression and cardiovascular events, including coronary artery disease, stroke, and heart failure (Barzilay JI et al., 2024).[9] Monitoring albuminuria allows for patient risk stratification, guiding treatment decisions and determining appropriate follow-up intervals, as reductions in albuminuria have been shown to correlate with slower CKD progression and a lower likelihood of progression to end-stage kidney disease.[10] Furthermore, a significant decrease in albuminuria, such as a 50% reduction, may indicate a favorable response to therapeutic interventions, including the use of renin-angiotensin system inhibitors, and is associated with a notable reduction in cardiovascular risk and heart failure incidence.[11] Conversely, a doubling of the albumin-to-creatinine ratio (ACR) on follow-up testing surpasses normal laboratory variability and has been associated with a higher incidence of CKD stage 4-5, thereby warranting further clinical evaluation.[12]

While albuminuria provides significant insight into kidney damage and its potential progression, it is equally important to consider other diagnostic markers, such as urine sediment abnormalities, which complement albuminuria in forming a more comprehensive picture of renal health. Urine sediment abnormalities refer to the presence of atypical elements in the urine, such as red and white blood cells, casts, crystals, and microorganisms, which are typically identified through microscopic examination after centrifugation. These abnormalities serve as an important diagnostic marker for CKD, as defined by the KDIGO 2024 Clinical Practice Guideline, which states that CKD is characterized by structural or functional kidney abnormalities persisting for more than three months. Analyzing urine sediment can help identify underlying causes, such as red blood cell casts indicating glomerulonephritis, white blood cell casts suggesting interstitial nephritis or pyelonephritis, and granular casts associated with acute tubular necrosis.[1]

This foundational understanding of urine sediment abnormalities naturally extends to the broader recognition of their diagnostic value in nephrology. Urine sediment analysis, microscopic examination of abnormal elements in urine such as blood cells, casts, crystals, and microorganisms, has long been regarded as a valuable diagnostic tool. Perazella (2015) highlights that this method remains an effective urinary biomarker, capable of detecting kidney disease and providing critical information about the specific compartment of renal injury.[13] Similarly, Cavanaugh (2019) emphasizes that urine sediment examination continues to serve as a classic, information-rich approach to kidney disease evaluation.[14] Furthermore, the American Family Physician (2017) clinical guideline recommends the use of urine sediment analysis when intrinsic kidney disease is suspected.[6]

Urine sediment analysis not only aids in discerning underlying renal pathologies but is also invaluable for diagnosis and prognosis in clinical practice. Indeed, Perazella (2015) underscores

that sediment examination "alerts the clinician to the presence of kidney disease and provides diagnostic information that often identifies the compartment of kidney injury".[13] Cavanaugh (2019) similarly maintains that it "remains a time-honored test that continues to provide substantial information about the patient's underlying kidney disease". Practical data further reinforce its utility: when nephrologists conduct urine sediment examinations versus automated lab analysis, they more accurately detect pathologic casts and dysmorphic red blood cells, achieving near-perfect diagnostic and prognostic accuracy for conditions like acute tubular injury and glomerulonephritis compared to a kidney biopsy.[15] These findings highlight that urine sediment assessment is a highly specific and powerful tool in the accurate diagnosis and longitudinal evaluation of CKD, justifying its role as an indispensable component of kidney disease work-ups.

Building upon the diagnostic strength of urine sediment analysis, a comprehensive evaluation of CKD also necessitates functional assessment, where estimated glomerular filtration rate (eGFR) plays a pivotal role. Estimated glomerular filtration rate (eGFR) is one of the critical markers in the detection, evaluation, and management of CKD. It is a calculated value that approximates the rate at which the kidneys filter waste from the blood, expressed in milliliters per minute per 1.73 square meters of body surface area (mL/min/1.73 m^2). It is derived from serum creatinine levels, age, sex, and ethnicity, providing a practical assessment of kidney function. eGFR aids in risk stratification for CKD progression and related complications.[1]

Table 2. Glomerular Filtration Rate (GFR) Grading[1]

GFR category	GFR (ml/min/per 1.73 m^2)	Terms
G1	≥90	Normal or high
G2	60-89	Mildly decreased
G3a	45-59	Mildly to moderately decreased
G3b	30-44	Moderately to severely decreased
G4	15-29	Severely decreased
G5	<15	Kidney failure

While the KDIGO 2024 guidelines recommend using externally validated risk equations to estimate the absolute risk of kidney failure in individuals with CKD stages G3–G5—such as identifying a 5-year kidney failure risk of 3%–5% to inform nephrology referral, eGFR and the urine albumin-to-creatinine ratio (ACR) remain foundational for both disease staging and management. eGFR influences treatment decisions such as initiating SGLT2 inhibitors in type 2 diabetes with CKD and eGFR ≥20 mL/min/1.73 m^2, while regular monitoring of eGFR is vital for tracking progression, assessing therapeutic efficacy, and adjusting care plans as needed.[1]k

While eGFR and ACR remain core parameters for staging and guiding the management of CKD, they are most effective when interpreted alongside other diagnostic indicators that reflect underlying renal pathology. Among these, persistent hematuria serves as an

early marker of glomerular injury, often preceding measurable changes in kidney function and adding further depth to risk stratification. Persistent hematuria represents one of the earliest clinical indicators of underlying glomerular disease, often preceding measurable declines in kidney function.[16] Evidence shows that even mild or moderate microscopic hematuria is associated with an increased risk of CKD progression and mortality, underscoring its diagnostic value in identifying subclinical renal injury and initiating early management strategies.[17]

Beyond glomerular involvement, tubular disorders may present with electrolyte and acid-base abnormalities such as hyperkalemia or metabolic acidosis, which reflect impaired tubular function. Research indicates that these disturbances not only serve as markers of renal damage but can also precede significant reductions in glomerular filtration rate (GFR), making early recognition crucial to slow disease progression through corrective interventions.[18]

Histopathological assessment through kidney biopsy remains the gold standard for diagnosing structural renal abnormalities, including interstitial fibrosis and glomerulosclerosis, which carry strong prognostic implications for CKD progression.[19] Complementing histological evaluation, non-invasive imaging modalities such as ultrasound, CT, and MRI provide critical insights into renal morphology, enabling detection of structural abnormalities like cystic disease or congenital malformations before overt functional decline.[20]

Furthermore, individuals with a history of kidney transplantation represent a unique population in which close surveillance for CKD markers is essential, given their elevated risk of graft dysfunction and recurrent disease. Early identification of abnormalities in this group allows timely therapeutic adjustments and improves long-term graft survival.[21]

Table 3. Pros and cons of kidney markers

Parameter	Pros	Cons
Albuminuria	- Strong predictor of CKD progression and cardiovascular events[22] - Cost-effective in targeted screening, low cost[23,24]	- Biological and analytic variability (exercise, fever, UTI) → repeat testing needed[1]
Urine sediment	- Detects casts/dysmorphic RBCs for localizing injury[25] - Recommended for suspected intrinsic disease[25] - Low cost[15]	- Operator-dependent[25]
eGFR	- Core for staging, drug dosing → primary marker for determining the stage of CKD and is used to adjust drug dosages to prevent toxicity in patients with impaired kidney function.[1] - Accuracy improves with cystatin C or 2021 CKD-EPI[26] - Cost: Low (creatinine)[27]	- Creatinine influenced by age, sex, muscle mass, diet, and tubular secretion[27] - Cost: Moderate (cystatin C)[28]

Electrolyte and acid-base abnormalities	- Reveal tubular dysfunction; low bicarbonate linked to faster CKD progression[29] - Cost: Low - moderate[29]	- This is a non-specific clinical and diagnostic sign that typically appears in the later stages of disease and cannot be used alone to establish a definitive diagnosis.[30]
Structural abnormalities	- USG: Detects obstruction, cysts, cortical changes[20] - CT/MRI: High sensitivity for masses, stones, and complex cysts[20] - Cost: USG: Low - moderate[20]	- USG: Operator - dependent[20] - CT/MRI: Radiation/contrast risk[20] - MRI: Often requires sedation[20] - Cost: Moderate - high (CT)[20] - Cost: High (MRI)[20]

Collectively, these diagnostic markers, including hematuria, tubular disorders, histological abnormalities, imaging findings, and post-transplant monitoring, establish a comprehensive framework for the early detection and longitudinal assessment of CKD. This integrated approach underscores the importance of combining conventional markers such as serum creatinine (SCr), estimated glomerular filtration rate (eGFR), and albuminuria with point-of-care testing (POCT), standardized laboratory assessments, and risk-prediction tools (e.g., QKidney, Kidney Health Australia Risk Test). Additional measurements, including urinary albumin-creatinine ratio (ACR), microalbuminuria (MAU), proteinuria, cystatin C, and emerging biomarkers, further enhance diagnostic precision, enabling timely interventions to preserve renal function and improve patient outcomes.[31]

EVALUATION WITH GFR, WHICH EQUATION WE SHOULD USE?

The accuracy of eGFR equations relies heavily on their input biomarkers. Creatinine, while cheap and ubiquitous, is flawed by its dependence on muscle mass, necessitating age and sex adjustments. It also suffers from a "blind spot" due to tubular secretion and susceptibility to drug interference.[1,32] Conversely, Cystatin C is biologically superior; produced constantly by all nucleated cells and independent of muscle mass, it offers greater accuracy for the elderly and those with altered body composition. Additionally, the difference between cystatin and creatinine estimates ("eGFRdiff") strongly predicts cardiovascular mortality.[33] However, widespread adoption is limited by high costs, restricting Cystatin C primarily to confirmatory testing.

For nearly five decades, the Cockcroft-Gault (C-G) equation underpinned pharmacokinetic dosing. However, its reliance on total body weight has rendered it dangerous in an era of rising obesity prevalence, as it systematically overestimates clearance in patients with high adiposity, leading to potential drug toxicity.[32] The 2024 FDA Guidance effectively ended the clinical relevance of C-G, recommending contemporary eGFR equations for drug development.[34] This shift paved the way for the MDRD and CKD-EPI 2009 equations, which standardized assays and introduced Body Surface Area (BSA) indexing. While the CKD-EPI 2009 equation became the global gold standard for its improved precision at higher GFRs, it retained an ethnic coefficient that adjusted estimates upward for Black patients, a feature that would eventually be challenged on ethical grounds.[35]

The definition of superiority underwent a radical re-evaluation with the 2021 reports from the NKF-ASN Task Force. Recognizing that ethnicity is a sociological construct rather than a biological determinant, the Task Force recommended the immediate adoption of the CKD-EPI 2021 Creatinine (ethnicity-free) Equation.[32] This transition was driven by the

imperative to eliminate systemic racism in medicine; the removal of the ethnic coefficient ensures that Black patients are not systematically disqualified from transplant waitlists due to artificially inflated eGFR values.[36] While this refitting resulted in a slight loss of statistical precision for non-Black populations—leading to minor overestimation of GFR—the nephrology community has largely accepted this trade-off, prioritizing health equity and the standardization of care over marginal statistical gains.[37] Studies indicate that approximately 45.8% of Black adults with CKD stages 3–5 would be reclassified to a more severe stage using the 2021 equation, directly impacting clinical management.[38]

While the US focused on equity, European researchers targeted the "age-gap" problem, the disjointed transition between pediatric and adult equations that disrupts longitudinal care. The European Kidney Function Consortium (EKFC) developed a novel equation utilizing a "Q-value" (median normal creatinine for age/sex) to create a seamless continuum from age 2 to over 90.[35] Validation studies suggest that the EKFC equation may be mathematically superior in European and East Asian cohorts, particularly in the elderly, where it avoids the overestimation bias seen with CKD-EPI.[39] The narrative of a "universal" equation fractures further when applied to Asian populations. The anthropometric differences in muscle mass-to-BSA ratios mean that Western-derived equations often fail in Japan, China, and South Asia. For instance, the CKD-EPI 2021 equation significantly overestimates GFR in Japanese populations, necessitating the use of the specific Japanese Society of Nephrology (JSN) equation to prevent the massive underdiagnosis of CKD.[1] Similarly, validation studies in Pakistan indicate that locally derived equations (PK-CKD-EPI) significantly outperform global models, underscoring that biological validation must precede clinical implementation in diverse ethnic groups.[40] In China, the BIS (Berlin Initiative Study) equation has shown promise for the elderly, further complicating the choice of a single standard.[41]

The comprehensive analysis of current literature indicates that no single equation holds universal superiority; rather, the "best" equation is contingent upon the clinical context. From a purely scientific perspective, the CKD-EPI 2021 Creatinine-Cystatin C Combined Equation is unequivocally the most accurate mathematical model. By integrating two biomarkers with disparate non-GFR determinants, it cancels out individual errors, yielding the highest P30 accuracy (>90%) and the most robust risk prediction.[42]

In the realm of public health, particularly within the United States, the CKD-EPI 2021 Creatinine (ethnicity-free) Equation reigns superior for routine screening. This approach successfully balances the operational requirement of using a low-cost biomarker with the ethical mandate to eliminate ethnic-based health disparities. Finally, regarding pharmacological applications, the BSA-Unindexed CKD-EPI 2021 equation has emerged as the superior method for drug dosing, replacing the flawed Cockcroft-Gault equation to ensure safer dosing in patients with extremes of body size.[34]

WHAT PRIMARY CARE CAN DO ABOUT CKD?

Early detection of CKD is essential to prevent disease progression and adverse outcomes. Screening should target individuals at elevated risk based on well-established predispositions. These include patients with diabetes mellitus (type 1 and 2), hypertension, cardiovascular disease, including a history of heart attack or stroke, obesity, autoimmune disorders like lupus or IgA nephropathy, history of recurrent kidney stones or chronic urinary tract infections (UTIs), and a family history of CKD or kidney failure. Additionally, individuals aged ≥ 60 years and those of African, South Asian, or Hispanic background carry a higher risk, as do those who are exposed to nephrotoxic agents, such as long-term NSAIDs, calcineurin inhibitors, or radiographic contrast, or environmental toxins like heavy metals or industrial solvents. Kidney transplant recipients also represent a high-risk group warranting

vigilant monitoring. Clinical indicators for evaluation include persistent hematuria or proteinuria, eGFR < 60 mL/min/1.73 m², uncontrolled hypertension, fatigue, and electrolyte or acid–base disturbances without alternative explanations (KDIGO 2024).[1,43–45]

Screening for these high-risk individuals involves periodic assessments of eGFR (preferably creatinine-based, with cystatin C if available) and urine albumin-to-creatinine ratio (uACR), backed by risk prediction tools such as QKidney or the Kidney Health Australia calculator to enhance five-year risk identification (KDIGO 2024).[1,31] A CKD diagnosis is confirmed when markers, such as albuminuria, urine sediment abnormalities, structural changes on imaging, or sustained eGFR decline, persist for at least three months.[1] Once diagnosed, CKD management requires a multifaceted approach: lifestyle modifications (healthy diet, exercise, weight control, and smoking cessation), blood pressure control with target <120/80 mm Hg using renin-angiotensin inhibitors, individualized glycemic targets for diabetes patients, and use of SGLT2 inhibitors (particularly in type 2 diabetes patients with eGFR ≥ 20 mL/min/1.73 m²) due to their renal and cardiovascular benefits.[1]

Ongoing monitoring of eGFR and uACR (at least annually or more frequently in high-risk cases) is critical. A greater-than 20% eGFR decline or a doubling of ACR between tests exceeds expected variability and should prompt further clinical action.[1] Referral to a nephrologist is warranted in cases of rapid progression, refractory hypertension, substantial albuminuria, or in anticipation of renal replacement therapy. Finally, patient education and shared decision-making empower individuals to understand CKD, adhere to treatment, and participate actively in their care, ultimately improving outcomes and preserving kidney function. By implementing these strategies, primary care physicians can significantly impact the early detection, effective management, and prevention of CKD progression, ultimately improving patient outcomes.

As for implementation in Indonesia, Cystatic C and uACR were not widely available. This test is considered vital for diagnosing and assessing the patient's current condition. A potential solution is to request the provision of this test in primary health care, supported by the government. However, for the Indonesian government to approve this, a valid cost-effectiveness study is certainly required. Therefore, a multicenter study involving health economists, public health experts, and nephrologists is vital to demonstrate the cost-effectiveness and long-term benefits of this test in slowing the progression of CKD cases. Whilst awaiting the results of the research, primary care facilities may refer these patients for routine monitoring of Cystatin C and uACR levels, as these are key parameters for monitoring the patient's condition. As general practitioners in healthcare settings, it is essential to understand the role and monitoring of Cystatin C and uACR, so that this data is not merely recorded but used to inform further patient management.

Conclusion

The KDIGO 2024 Clinical Practice Guideline marks a crucial inflection point in CKD management, synthesizing a decade of evidence into an integrated, ethically-driven framework. A key finding is the imperative to adopt a multi-marker approach for diagnosis, moving beyond sole reliance on Glomerular Filtration Rate (GFR) to include persistent markers of kidney damage such as albuminuria and urine sediment abnormalities. For functional assessment, the guidelines strongly advocate for the ethnicity-free CKD-EPI 2021 equation for routine screening to advance health equity, and the combined Creatinine-Cystatin C equation for superior accuracy when available. Pharmacologically, the strong recommendation for SGLT2 inhibitors in Type 2 Diabetes patients with CKD introduces a potent therapeutic tool for renoprotection. This review directly addresses the critical research gap concerning the primary care sector's capacity and capability to implement these global standards. We conclude that primary care, as the indispensable frontline of health

services, must rapidly assimilate these KDIGO 2024 updates. By doing so, primary care physicians can significantly enhance early screening, refine patient risk stratification, and ensure timely, informed referrals to specialist care, thereby effectively mitigating disease progression and improving patient outcomes in high-burden, resource-limited settings globally.

Declarations

Competing interests

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Author's Contribution

Idea/concept: AMS, LPS. Design: AMS, SYH, LPS. Control/supervision: LPS. Data collection/ processing: AMS, SYH. Analysis/ interpretation: AMS, SYH. Literature review: AMS, SYH. Writing the article: AMS, SYH. Critical review: LPS. All authors have critically reviewed and approved the final draft and are responsible for the content and similarity index of the manuscript.

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