Executive summary of the KDIGO 2024 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease: known knowns and known unknowns

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The Kidney Disease: Improving Global Outcomes (KDIGO) Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease (CKD) updates the KDIGO 2012 guideline and has been developed with patient partners, clinicians, and researchers around the world, using robust methodology. This update, based on a substantially broader base of evidence than has previously been available, reflects an exciting time in nephrology. New therapies and strategies have been tested in large and diverse populations that help to inform care; however, this guideline is not intended for people receiving dialysis nor those who have a kidney transplant. The document is sensitive to international considerations, CKD across the lifespan, and discusses special considerations in implementation. The scope includes chapters dedicated to the evaluation and risk assessment of people with CKD, management to delay CKD progression and its complications, medication management and drug stewardship in CKD, and optimal models of CKD care. Treatment approaches and actionable guideline recommendations are based on systematic reviews of relevant studies and appraisal of the quality of the evidence and the strength of recommendations which followed the "Grading of Recommendations Assessment, Development, and Evaluation" (GRADE) approach. The limitations of the evidence are discussed. The guideline also provides practice

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points, which serve to direct clinical care or activities for which a systematic review was not conducted, and it includes useful infographics and describes an important research agenda for the future. It targets a broad audience of people with CKD and their healthcare, while being mindful of implications for policy and payment.

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This 2024 update of the Kidney Disease: Improving Global Outcomes (KDIGO) Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease (CKD) heralds a new era in the care of people with kidney diseases. The majority of statements from the 2012 guideline have been updated based on current knowledge and practice. Only 6 statements were retained in their original form in 2012.

There is clear and increasing recognition of CKD as a global public health problem. The inclusion of people with CKD in clinical trials has improved substantially, thus generating an evidence base upon which to recommend care and treatments that have not previously existed. There are increasing efforts to improve diagnostic evaluation of cause, with increased sophistication of imaging methods, biopsy interrogation, and genetic evaluation, as well as methods to optimize blood and urine testing. With advances in technology, such as molecular diagnostics for tissue samples, integrated omics platforms, and the use of machine learning/artificial intelligence to explore large databases of both clinical and biological data, we are truly at the beginning of a new era in nephrology.

This guideline integrates existing and new knowledge to guide the care of people with CKD. It has been developed by an international Work Group that included patient partners, clinicians, and researchers with diverse experience across the spectrum of populations, a dedicated Evidence Review Team, and professional KDIGO staff. This clinical practice guideline includes 2 different types of statements: graded recommendations, which are supported by systematic reviews (i.e., *de novo* reviews conducted by the independent Evidence Review Team or existing high-quality reviews that have been systematically identified), and ungraded practice points, which serve to direct clinical care or activities for which a systematic review was not conducted for various reasons (e.g., lack of a sufficient evidence base or randomized controlled trials that would be impractical/unethical). Both recommendations and practice points are intended to help guide clinical practice and aid in decision-making; thus, collectively are the guideline statements. They are clearly articulated, actionable, and presented together so that all guideline statements can be implemented. The distinction between them is based on the process by which they are derived, and that process is based on the framework methodology from the KDIGO Methods Committee and aligns with other international guideline groups using the "Grading of Recommendations Assessment, Development, and Evaluation" (GRADE) methodology.

New developments in the refinement of evaluation of glomerular filtration rate (GFR), population and individual risk prediction, and novel treatments have all positively influenced the prognosis for people with CKD and are presented here. The Work Group has aimed to generate a guideline that is both rigorously devoted to new and existing evidence, and that is clinically useful. Research recommendations are presented in a separate section of the document and are intended to guide the next set of important research questions to inform and improve the outcomes of people living with CKD. We specifically urge the community to be inclusive of people across the lifecycle and include sex (referring to biological factors including genetics, sex steroids, physiology, and anatomy), gender (referring to sociocultural factors such as identity, roles, and relations), and etiology of CKD as important variables in all studies.

We offer recommendations to clinicians and clinical laboratories to understand and promote the standardization and accuracy of testing tools including assays and equipment. The effective use of clinical practice guidelines and, therefore, effective patient care, including accurate diagnosis and referral prioritization, clinical research, and public health prioritization, requires comparability of laboratory results independent of time, place, and measurement procedure. Key to this is establishing precision of testing and between-laboratory agreement with traceability to accepted international reference standards wherever available. Therefore, this guidance document includes standards for laboratory tests. Specifically, we focus on creatinine and cystatin C, with the goal of normalizing access to both tests for increased accuracy of GFR assessment, and the assessment of urine albumin which is also critical to risk assessment and care plans.

The guideline is organized into 6 chapters (Tables 1, 4, and 5 cover Chapters 1–5). In this summary, we outline the key evidence-based recommendations together with selected practice points by chapter. Readers are referred to the full guideline for a comprehensive description of benefits and harms, certainty of evidence, values and preferences, resource use and costs, factors affecting implementation, special considerations, and both general and specific research recommendations.

Qualifying statements, key concepts, special considerations, Chronic Kidney Disease Prognosis Consortium (CKD-PC)

Clarifying definition and classification. We begin with acknowledgment that the definition and classification system is widely accepted by the community. Specifically, we remind readers of the difference between the definition of CKD, which is inclusive of various markers of kidney damage, and the classification system that highlights the importance of the CGA system (i.e., **C**ause/**G**lomerular filtration rate level/**A**lbuminuria level) for the purposes of management, treatment, risk assessment, and research. The relative risk of many outcomes (CKD progression, kidney failure, acute kidney injury, infection,

hospitalizations, cardiovascular mortality, myocardial infarction, atrial fibrillation, stroke, and peripheral vascular disease) is increased for all people with CKD, whereas absolute risks for individuals are modified by age, sex, and other factors. We highlight the difference between relative and absolute risks (the latter derived by applying individual risk prediction scores), acknowledge that there are different risks for different populations, and do not support any age-adjusted definitions of CKD, as there are no age-adjusted definitions for diabetes, cardiovascular disease (CVD), nor hypertension, but rather recognize that the individual implications of those conditions for individuals differ by age group.

Screening. Despite the increasing recognition of the true burden of CKD, there remains controversy and lack of consensus as to the utility of population screening for CKD[1] or targeted screening programs[2] due to the complexity of the underlying sociopolitical and resource environment. Public health policy has a role to play in identifying and addressing risk factors to prevent CKD, to identify CKD early, and to delay its progression and associated adverse outcomes. Incorporating evidence-based treatment of people with CKD with sodium-glucose cotransporter-2 (SGLT2) inhibitors, together with a systematic review in people with diabetes and hypertension, suggests that screening adults for CKD could now be cost-effective.[3,4]

International considerations. In low- and middle-income countries and in the lower sociodemographic quintiles around the world, there is a large gap between CKD burden and provision of adequate healthcare. There is limited access to kidney replacement therapy combined with the rising prevalence of diabetes and hypertension and evidence of substantial sex and gender disparities in access to CKD treatment. Importantly, slowing CKD progression at early stages should provide economic benefits and prevent the development of kidney failure and cardiovascular complications. A systematic review of care models in low- and middle-income countries found that those supporting primary care providers or allied health workers achieved effectiveness in slowing GFR decline, as opposed to interventions centered on specialty care alone.[5] Where there are resource limitations, it is logical to deploy resources where they will be most cost-effective, for example, to higher-risk, preventable stages.

Special considerations and updated population data. We recognize that kidney diseases affect people at different times and with different impacts across the whole lifespan. Thus, enabling a personalized approach, considering age, sex, and gender for diagnosis, risk assessment, and treatment, is critical. At the extremes of age, the very young and the very old, diagnostic procedures, treatment aims, treatment modalities, and decision-making differ due to differences in prognosis, treatment options, and prioritization. In young and middle-aged adults, treatment approaches may differ because of specific circumstances, such as pregnancy or menopause. Sex (biological attributes) and gender (sociocultural factors), as well as other important intersectional factors, including, but not limited to, geographical location, socioeconomic position, and race/ethnicity, play important roles in kidney health and disease. Within the guideline document, we highlight concepts as to why age, sex, and gender should be considered, and how these specifically impact within each of the chapters (see Figure 1).

Multinational population studies, assessing risks, based on CKD-PC 2023 analyses,[6] are presented as part of the introductory chapter to further highlight updated information demonstrating epidemiological risk across CKD categories on a population level. The data describe that the association of all

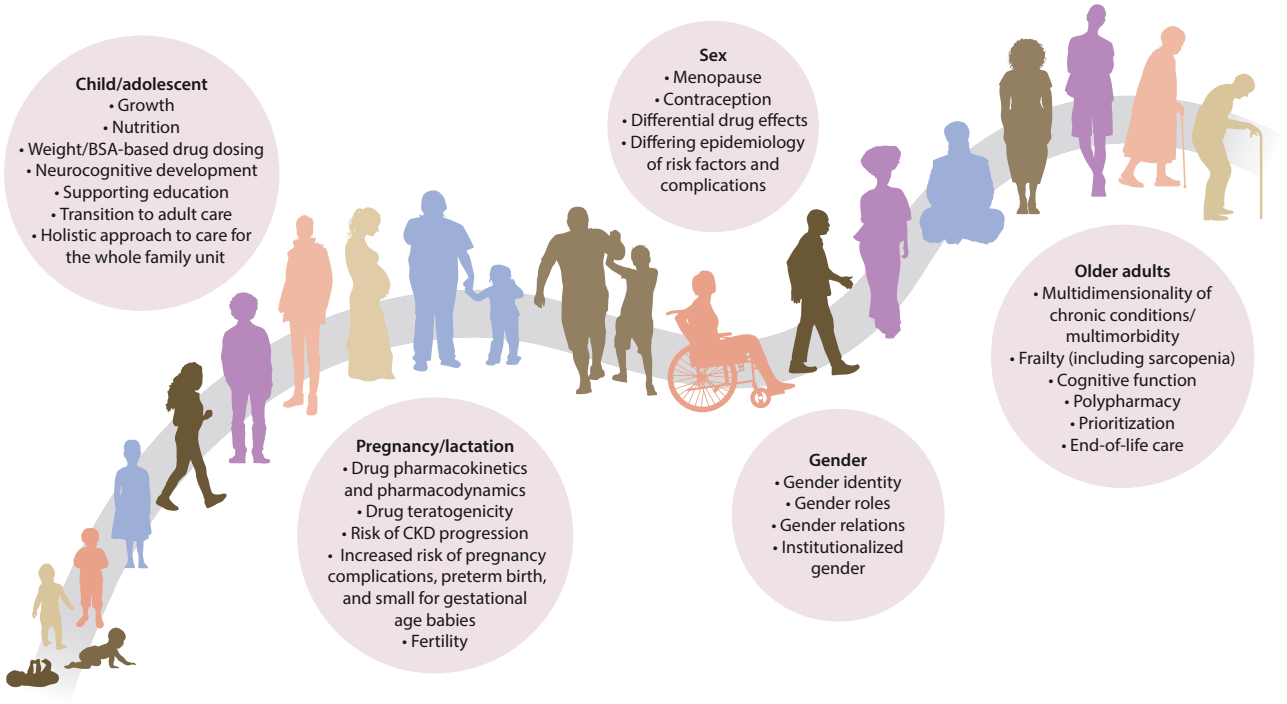

Figure 1 | Special considerations for chronic kidney disease (CKD) care across the lifespan. BSA, body surface area.

Table 1 | Recommendations and practice points from Chapters 1 and 2 of the KDIGO 2024 Clinical Practice Guideline for Evaluation and Management of CKD

Chapter 1. Evaluation of CKD

1.1 Detection and evaluation of CKD

1.1.1 Detection of CKD

Practice Point 1.1.1.1: Test people at risk for and with chronic kidney disease (CKD) using both urine albumin measurement and assessment of glomerular filtration rate (GFR).

Practice Point 1.1.1.2: Following incidental detection of elevated urinary albumin-to-creatinine ratio (ACR), hematuria, or low estimated GFR (eGFR), repeat tests to confirm presence of CKD.

1.1.2 Methods for staging of CKD

Recommendation 1.1.2.1: In adults at risk for CKD, we recommend using creatinine-based estimated glomerular filtration rate (eGFRcr). If cystatin C is available, the GFR category should be estimated from the combination of creatinine and cystatin C (creatinine and cystatin C–based estimated glomerular filtration rate [eGFRcr-cys]) (*1B*).

1.1.3 Evaluation of chronicity

Practice Point 1.1.3.1: Proof of chronicity (duration of a minimum of 3 months) can be established by:

(i) review of past measurements/estimations of GFR;
(ii) review of past measurements of albuminuria or proteinuria and urine microscopic examinations;
(iii) imaging findings such as reduced kidney size and reduction in cortical thickness;
(iv) kidney pathological findings such as fibrosis and atrophy;
(v) medical history, especially conditions known to cause or contribute to CKD;
(vi) repeat measurements within and beyond the 3-month point.

Practice Point 1.1.3.2: Do not assume chronicity based upon a single abnormal level for eGFR and ACR, as the finding could be the result of a recent acute kidney injury (AKI) event or acute kidney disease (AKD).

Practice Point 1.1.3.3: Consider initiation of treatments for CKD at first presentation of decreased GFR or elevated ACR if CKD is deemed likely due to presence of other clinical indicators.

1.1.4 Evaluation of cause

Practice Point 1.1.4.1: Establish the cause of CKD using clinical context, personal and family history, social and environmental factors, medications, physical examination, laboratory measures, imaging, and genetic and pathologic diagnosis (Figure 8[a]).

Practice Point 1.1.4.2: Use tests to establish a cause based on resources available (Table 6[b]).

Recommendation 1.1.4.1: We suggest performing a kidney biopsy as an acceptable, safe, diagnostic test to evaluate cause and guide treatment decisions when clinically appropriate (*2D*).

1.2 Evaluation of GFR

1.2.1 Other functions of kidneys besides GFR

Practice Point 1.2.1.1: Use the term "GFR" when referring to the specific kidney function of glomerular filtration. Use the more general term "kidney function(s)" when dealing with the totality of functions of the kidney.

1.2.2 Guidance to physicians and other healthcare providers

Practice Point 1.2.2.1: Use serum creatinine (SCr) and an estimating equation for initial assessment of GFR (Figure 11[c]).

Recommendation 1.2.2.1: We recommend using eGFRcr-cys in clinical situations when eGFRcr is less accurate and GFR affects clinical decision-making (Table 8[d]) (*1C*).

Practice Point 1.2.2.2: Where more accurate ascertainment of GFR will impact treatment decisions, measure GFR using plasma or urinary clearance of an exogenous filtration marker (Table 9[e]).

Practice Point 1.2.2.3: Understand the value and limitations in both eGFR and measured glomerular filtration rate (mGFR) as well as the variability and factors that influence SCr and cystatin C measurements.

Practice Point 1.2.2.4: Interpretation of SCr level requires consideration of dietary intake.

Practice Point 1.2.2.5: Assess the potential for error in eGFR when assessing a change in GFR over time.

Practice Point 1.2.2.6: Consider the use of cystatin C–based estimated glomerular filtration rate (eGFRcys) in some specific circumstances.

Practice Point 1.2.2.7: Understand the implications of differences between eGFRcr and eGFRcys, as these may be informative, in both direction and magnitude of those differences.

Practice Point 1.2.2.8: Consider timed urine collections for measured creatinine clearance if mGFR is not available and eGFRcr-cys is thought to be inaccurate.

1.2.3 Guidance to clinical laboratories

Practice Point 1.2.3.1: Implement the laboratory standards of care outlined in Table 11[f] to ensure accuracy and reliability when assessing GFR using creatinine and cystatin C.

Practice Point 1.2.3.2: Given available resources, clinical laboratories may consider the possibility of measurement of both creatinine and cystatin either as an in-house test or as a referred test.

Special considerations

Pediatric considerations

Practice Point 1.2.3.3: Laboratories measuring creatinine in infants or small children must ensure their quality control process include the lowest end of the expected range of values for the group of interest.

(*Continued on following page*)

Table 1 | (Continued) **Recommendations and practice points from Chapters 1 and 2 of the KDIGO 2024 Clinical Practice Guideline for Evaluation and Management of CKD**

Chapter 1. Evaluation of CKD

Practice Point 1.2.3.4: Consider the consistent use of enzymatic creatinine assays in children, given the higher relative contribution of non-creatinine chromogens to measured creatinine in children when using the Jaffe assay, and the high prevalence of icteric and hemolyzed samples in the neonatal period.

Practice Point 1.2.3.5: An eGFRcr level <90 ml/min per 1.73 m^2 can be flagged as "low" in children and adolescents over the age of 2 years.

1.2.4 Selection of GFR estimating equations

Recommendation 1.2.4.1: We recommend using a validated GFR estimating equation to derive GFR from serum filtration markers (eGFR) rather than relying on the serum filtration markers alone (*1D*).

Practice Point 1.2.4.1: Use the same equation within geographical regions (as defined locally [e.g., continent, country, and region] and as large as possible). Within such regions, equations may differ for adults and children.

Practice Point 1.2.4.2: Use of race in the computation of eGFR should be avoided.

Special considerations

Pediatric considerations

Practice Point 1.2.4.3: Estimate GFR in children using validated equations that have been developed or validated in comparable populations.

1.3 Evaluation of albuminuria

1.3.1 Guidance for physicians and other healthcare providers

Practice Point 1.3.1.1: Use the following measurements for initial testing of albuminuria (in descending order of preference). In all cases, a first void in the morning midstream sample is preferred in adults and children.

(i) urine ACR, or
(ii) reagent strip urinalysis for albumin and ACR with automated reading.

If measuring urine protein, use the following measurements:

(i) urine protein-to-creatinine ratio (PCR),
(ii) reagent strip urinalysis for total protein with automated reading, or
(iii) reagent strip urinalysis for total protein with manual reading.

Practice Point 1.3.1.2: Use more accurate methods when albuminuria is detected using less accurate methods.

- Confirm reagent strip positive albuminuria and/or proteinuria by quantitative laboratory measurement and express as a ratio to urine creatinine wherever possible (i.e., quantify the ACR or PCR if initial semiquantitative tests are positive).
- Confirm ACR ≥30 mg/g (≥3 mg/mmol) on a random untimed urine with a subsequent first morning void in the morning midstream urine sample.

Practice Point 1.3.1.3: Understand factors that may affect interpretation of measurements of urine albumin and urine creatinine and order confirmatory tests as indicated (Table 16[g]).

Special considerations

Pediatric considerations

Practice Point 1.3.1.4: In children, obtain a first morning urine sample for initial testing of albuminuria and proteinuria (in descending order of preference):

(i) Both urine PCR and urine ACR,
(ii) Reagent strip urinalysis for total protein and for albumin with automated reading, or
(iii) Reagent strip urinalysis for total protein and for albumin with manual reading.

1.3.2 Guidance to clinical laboratories

Practice Point 1.3.2.1: Implement the laboratory reporting and handling standards outlined in Table 17[h] to ensure accuracy and reliability of the findings when assessing urine samples.

Practice Point 1.3.2.2: Implementation of an external quality assessment scheme/program for urine albumin and creatinine, including calculation of the ACR, is a preferred practice for laboratories.

1.4 Point-of-care testing

Recommendation 1.4.1: We suggest that point-of-care testing (POCT) may be used for creatinine and urine albumin measurement where access to a laboratory is limited or providing a test at the point-of-care facilitates the clinical pathway (*2C*).

Practice Point 1.4.1: Whenever a POCT device is used for creatinine and urine albumin testing, ensure that the same preanalytical, analytical, and postanalytical quality criteria relating to the specimen collection and performance of the device, including external quality assessment, and the interpretation of the result is used.

Practice Point 1.4.2: Where a POCT device for creatinine testing is being used, generate an estimate of GFR. Use the equation consistent with that used within the region.

Practice Point 1.4.3: Where a POCT device is being used for albuminuria testing, the capability of also analyzing creatinine and producing an ACR is important. Assess the ability of the POCT ACR devices to produce a positive result in 85% of people with significant albuminuria (ACR ≥30 mg/g or ≥3 mg/mmol), as part of the evaluation and consideration of using the device.

Chapter 2. Risk assessment in people with CKD

2.1 Overview on monitoring for progression of CKD based upon GFR and ACR categories

Practice Point 2.1.1: Assess albuminuria in adults, or albuminuria/proteinuria in children, and GFR at least annually in people with CKD.

Practice Point 2.1.2: Assess albuminuria and GFR more often for individuals at higher risk of CKD progression when measurement will impact therapeutic decisions.

Practice Point 2.1.3: For people with CKD, a change in eGFR of >20% on a subsequent test exceeds the expected variability and warrants evaluation.

Practice Point 2.1.4: Among people with CKD who initiate hemodynamically active therapies, GFR reductions of >30% on subsequent testing exceed the expected variability and warrant evaluation.

(*Continued on following page*)

Table 1 | (Continued)

Chapter 2. Risk assessment in people with CKD
Practice Point 2.1.5: For albuminuria monitoring of people with CKD, a doubling of the ACR on a subsequent test exceeds laboratory variability and warrants evaluation.
2.2 Risk prediction in people with CKD
Recommendation 2.2.1: In people with CKD G3–G5, we recommend using an externally validated risk equation to estimate the absolute risk of kidney failure (*1A*).
Practice Point 2.2.1: A 5-year kidney failure risk of 3%–5% can be used to determine need for nephrology referral in addition to criteria based on eGFR or urine ACR, and other clinical considerations.
Practice Point 2.2.2: A 2-year kidney failure risk of >10% can be used to determine the timing of multidisciplinary care in addition to eGFR-based criteria and other clinical considerations.
Practice Point 2.2.3: A 2-year kidney failure risk threshold of >40% can be used to determine the modality education, timing of preparation for kidney replacement therapy (KRT) including vascular access planning or referral for transplantation, in addition to eGFR-based criteria and other clinical considerations.
Practice Point 2.2.4: Note that risk prediction equations developed for use in people with CKD G3–G5, may not be valid for use in those with CKD G1–G2.
Practice Point 2.2.5: Use disease-specific, externally validated prediction equations in people with immunoglobulin A nephropathy (IgAN) and autosomal dominant polycystic kidney disease (ADPKD).
2.3 Prediction of cardiovascular risk in people with CKD
Practice Point 2.3.1: For cardiovascular risk prediction to guide preventive therapies in people with CKD, use externally validated models that are either developed within CKD populations or that incorporate eGFR and albuminuria.
Practice Point 2.3.2: For mortality risk prediction to guide discussions about goals of care, use externally validated models that predict all-cause mortality specifically developed in the CKD population.

CKD, chronic kidney disease; KDIGO, Kidney Disease: Improving Global Outcomes.
[a]Figure 8 in full guideline.
[b]Table 6 in full guideline.
[c]Figure 11 in full guideline.
[d]Table 8 in full guideline.
[e]Table 9 in full guideline.
[f]Table 11 in full guideline.
[g]Table 16 in full guideline.
[h]Table 17 in full guideline.

complications of CKD is incrementally increased with worse categories of estimated GFR (eGFR) and albuminuria and include information using both updated eGFR equations for creatinine alone, as well as those using creatinine and cystatin C combined.

On this foundation of key concepts and inclusive framework, we now describe the key statements for each of the chapters.

Chapter 1: Evaluation of CKD

This chapter highlights the importance of detecting CKD in high-risk populations because CKD is often silent and patients may be asymptomatic at early stages. The screening algorithm for the diagnosis of CKD in adults highlights the use of basic testing methods, and then details the importance of thorough evaluation for the cause and accurate assessment of GFR and urine albumin-to-creatinine ratio (ACR) to appropriately stage people with CKD. There are special considerations in pediatric populations and those with advanced age regarding screening and staging. The evaluation of cause should be thorough, using all available information (clinical, social, and family history) and accessible testing (laboratory, imaging, genetics, and biopsy), given that etiology impacts prognosis, risk, and choice of therapies. Given that up to 25% of all current CKD registries or study cohorts have "unknown etiology" documented, there is renewed interest in enabling clinicians and people with CKD to understand, if possible, the cause of CKD.

The chapter also reminds clinicians that the kidney has many functions (excretory, endocrine, and metabolic) and that GFR is one component of excretory function. Understanding the best methods by which to evaluate GFR and when to use estimating equations versus direct measurements is highlighted.

The recommendations to use the most accurate, validated equations to estimate GFR, preferably with a combination of serum creatinine and cystatin C, are important. From both a patient and provider perspective, acknowledging that serum creatinine–based equations have limitations that can be overcome with the use of an additional marker (cystatin C) is notable and places a high value on accuracy and lower value on resource utilization associated with the additional biomarker. The chapter describes the value of using a combined creatinine and cystatin C–based equation in specific situations. Table 2 highlights clinical circumstances where this might apply.[7–23] As we acquire more knowledge and data about GFR estimating equations using cystatin C versus creatinine or both, there are now recognized implications of those differences that need to be understood by clinicians, in both direction and magnitude.

There are issues related to how best to estimate GFR in transgender, gender-diverse, or nonbinary individuals where a

Table 2 | Indications for use of cystatin C

Domain	Specific clinical condition	Cause of decreased accuracy	Comments on GFR evaluation
Body habitus and changes in muscle mass	Eating disorders[7]	Non-GFR determinants of SCr	eGFRcys may be appropriate if no comorbid illness other than reduction in muscle mass.
	Extreme sport/exercise/body builder	Non-GFR determinants of SCr	eGFRcys may be appropriate if an increase in muscle mass is the only abnormality.
	Above-knee amputation[8]	Non-GFR determinants of SCr	eGFRcys may be appropriate in those without other comorbid conditions. Suggest eGFRcr-cys in those with comorbid illness.
	Spinal cord injury with paraplegia/ paraparesis or quadriplegia/ quadriparesis	Non-GFR determinants of SCr	eGFRcys may be appropriate in those without other comorbid illness. Suggest eGFRcr-cys in those with no comorbid illness.
	Class III obesity[a,b]	Non-GFR determinants of SCr and SCys	eGFRcr-cys demonstrated to be most accurate.
Lifestyle	Smoking[9–11]	Non-GFR determinants of SCys	Minimal data, suggest eGFRcr if no changes to non-GFR determinants of SCr or comorbid illness.
Diet	Low-protein diet	Non-GFR determinants of SCr	Minimal data, suggest eGFRcr may be appropriate if no changes to non-GFR determinants of SCr or no comorbid illness.
	Keto diets	Non-GFR determinants of SCr	
	Vegetarian	Non-GFR determinants of SCr	
	High-protein diets and creatine supplements	Non-GFR determinants of SCr	
Illness other than CKD	Malnutrition	Chronic illness, presumed impact on non-GFR determinants of SCr and SCys	eGFRcr-cys may be less accurate because of coexistence of malnutrition and inflammation. Suggest using mGFR for treatment decisions based on the level of GFR.
	Cancer[a,12-16]	Chronic illness, presumed impact on non-GFR determinants of SCr and SCys	eGFRcr-cys demonstrated to be most accurate in populations studied but likelihood of lesser accuracy in more frail people or in cancers with high cell turnover. Suggest using mGFR for treatment decisions based on the level of GFR.
	Heart failure[a,17,18]	Chronic illness, presumed impact on non-GFR determinants of SCr and SCys	Although limited data, eGFRcys appears less biased but all have low accuracy. Suggest using eGFRcr-cys or eGFRcys for routine GFR evaluation. Suggest using mGFR for treatment decisions based on the level of GFR.
	Cirrhosis[a,19-21]	Chronic illness, presumed impact on non-GFR determinants of SCr and SCys	Although limited data, eGFRcys appears less biased but all have low accuracy. Suggest using eGFRcr-cys or eGFRcys for routine GFR evaluation. Suggest using mGFR for treatment decisions based on the level of GFR.
	Catabolic consuming diseases[c]	Chronic illness, presumed impact on non-GFR determinants of SCr and SCys	Minimal data but eGFRcr-cys may be inaccurate. Suggest using eGFRcr-cys vs. eGFRcr for routine GFR evaluation. Suggest using mGFR for treatment decisions based on the level of GFR.
	Muscle-wasting diseases[22]	Chronic illness, presumed impact on non-GFR determinants of SCr and SCys	Minimal data. One study shows large bias for both eGFRcr and eGFRcys. Suggest using eGFRcr-cys for routine GFR evaluation. Suggest using mGFR for treatment decisions based on the level of GFR.
Medication effects	Steroids (anabolic, hormone)	Non-GFR determinants of SCr. Effect on SCys not known	Physiological effect on SCys unknown, suggest eGFRcr-cys.
	Decreases in tubular secretion	Non-GFR determinants of SCr	eGFRcys may be appropriate if medication affects only creatinine and no comorbid illness. Suggest using mGFR for treatment decisions based on the level of GFR.
	Broad spectrum antibiotics that decrease extrarenal elimination	Non-GFR determinants of SCr	eGFRcys may be appropriate if medication affects only creatinine and no comorbid illness. Suggest using mGFR for treatment decisions based on the level of GFR.

eGFR, estimated glomerular filtration rate; eGFRcr, creatinine-based estimated GFR; eGFRcr-cys, creatinine and cystatin C–based estimated GFR; mGFR, measured GFR; SCr, serum creatinine; SCys, serum cystatin C.
[a]Data summarized in Adingwupu OM, Barbosa ER, Palevsky PM, et al. Cystatin C as a GFR estimation marker in acute and chronic illness: a systematic review. *Kidney Med*. 2023;5:100727.[23]
[b]Obesity class III varies by region but commonly body mass index >40 or >35 kg/m^2.
[c]Catabolic consuming disease may include tuberculosis, AIDS, hematologic malignancies, and severe skin diseases. There are no data with mGFR to evaluate this directly.

Table 3 | Indications for measured glomerular filtration rate

Clinical conditions in which eGFRcr-cys is inaccurate or uncertain due to potential non-GFR determinants of creatinine and cystatin C. This may include catabolic states, such as serious infections or inflammatory states, high cell turnover as in some cancers, advanced cirrhosis or heart failure, use of high-dose steroids, or the very frail. See Figure 12 in the full guideline for approach to individual decision-making.
Clinical settings in which greater accuracy is needed than is achieved with eGFRcr-cys. For example, decisions about simultaneous kidney transplant at the time of other solid organ transplant, kidney donor candidacy, and drug dosing if narrow therapeutic index or serious toxicity (e.g., chemotherapies that are cleared by the kidney).

eGFRcr-cys, creatinine and cystatin C–based estimated GFR; GFR, glomerular filtration rate.

person's gender identity is different from their sex assigned at birth, and they may or may not be taking gender hormone–affirming therapy or puberty-blocking therapies. The calculation of estimated GFR by creatinine and cystatin C using either sex constant may be impacted by non-GFR determinants of creatinine, and a holistic approach should be taken to determine appropriate management anchored to the muscle mass of the individual and based on their sex hormone configuration and gender identity. Similarly, cystatin C–based equations may be affected by gender hormone–affirming therapy. The assessment of gender in the context of eGFR is therefore an area for shared decision-making and an evolving area for investigation.

Where more accurate ascertainment of GFR will impact treatment decisions (e.g., oncology drug dosing, kidney donor eligibility, etc.), clinicians are encouraged to measure GFR by established clearance methods of glomerular filtration markers (Table 3).

The chapter addresses and reinforces key points for clinical laboratories including the implementation of laboratory standards for reporting, the use of enzymatic assays instead of Jaffe method for creatinine measurements (given the interference of many drugs and substances when using the latter), timing of processing of blood samples, and simultaneous measurement of creatinine and cystatin C on the same blood sample. These are all intended to improve consistency and comparability of laboratory values.

The chapter also recommends using validated GFR estimating equations that should be the same within geographical regions so that people with CKD and healthcare providers (HCPs) have access to the same information, derived using the same methodology.

The evaluation of albuminuria is also addressed in this chapter, so that clinicians are aware and understand the determinants of albuminuria measurements, which will enable better interpretation and guide action. Given the established, consistent strong relationship between the quantity of urine albumin with both kidney disease and CVD risk, clinicians are encouraged to pay attention to this measurement. Albuminuria measurements may not be available in all regions, and proteinuria is not the same as albuminuria; clinicians should appreciate those differences. There are factors that influence variability in urine albumin or protein measures, as there are different ones that impact urine creatinine. Given that the ratios are used to ascertain risk, understanding sources of both analytical and biological variability is important for the interpretation of fluctuations in urine ACR.

The use of point-of-care testing may facilitate access to earlier diagnosis and care and can be implemented in rural and remote locations. The value of point-of-care testing for currently underserved populations cannot be overstated and should include the capacity for generating creatinine-based eGFR equations. The point-of-care testing devices used would ideally measure both blood creatinine and urine for albumin and creatinine to measure ACR and be standardized and calibrated with similar rigor as is recommended for laboratory tests.

Overall, this chapter presents statements that help HCPs to fully evaluate people with the diagnosis of CKD and provides data to aid the clinicians and the laboratories to understand and appreciate nuances of evaluating the common tests for assessing GFR and albuminuria, and aims to aid in increasing accuracy of risk assessments to guide therapeutic decision-making.

Chapter 2: Risk assessment in people with CKD

This chapter addresses the need to monitor the progression of CKD using both blood and urine tests on a regular basis, informed by risk of the individual, and contextualized within healthcare system capacity. It provides guidance for both monitoring and the interpretation of changes over time that may prompt additional testing or evaluation. There is an expected variability in GFR and urine ACR caused by both biological and analytical factors of the biomarkers used, and nuances regarding changes with the initiation of therapies and when/how to further evaluate are also offered.

Importantly, this chapter recommends the use of validated risk equations to estimate the "absolute" risk of kidney failure for individual people and offers thresholds by which to determine referral timing of multidisciplinary care, modality education, and preparation for transition to kidney replacement therapy, either dialysis or transplantation. The differentiation of "relative risks" as presented in the heatmaps in the introductory chapter and individual risks, as calculated using these risk equations, is critical in the context of improving communication between people with CKD and HCPs, as well as planning for transitions in care, healthcare system resource utilization, and potentially for enrollment into clinical trials. It is noted that there are disease-specific tools (for immunoglobulin A nephropathy and polycystic kidney disease) and evolving tools for predicting events and mortality in people with CKD. In the era of precision medicine, the use of validated prediction tools will help tailor the frequency of visits, bloodwork, timing of educational

activities, and may inform a better selection of targets of care to support people and families living with CKD.

Chapter 3: Delaying CKD progression and managing its complications

Chapter 3 provides a comprehensive discussion of the totality of treatment strategies required to reduce the risk of both progression of CKD and its attendant comorbidities (Table 4).

Lifestyle factors that include optimizing physical activity and weight, the avoidance of tobacco products, as well as access to trained individuals (renal dietitians or accredited nutrition providers, pharmacists, psychologists, and others) are highlighted, with the evidence to support those activities presented. It is noted that those with specific needs (children or frail [older] adults) should have lifestyle counseling based on capability and values.

There is an extensive review of diets now recommended for people with CKD, including plant-based diets, and protein intake in accordance with needs and status. The importance of not restricting protein in those who are cachexic, sarcopenic, or undernourished is highlighted. Avoidance of high-protein intake and the need for close supervision if very low–protein intake is attempted are all described in detail, again, underscoring the importance of individualization of care

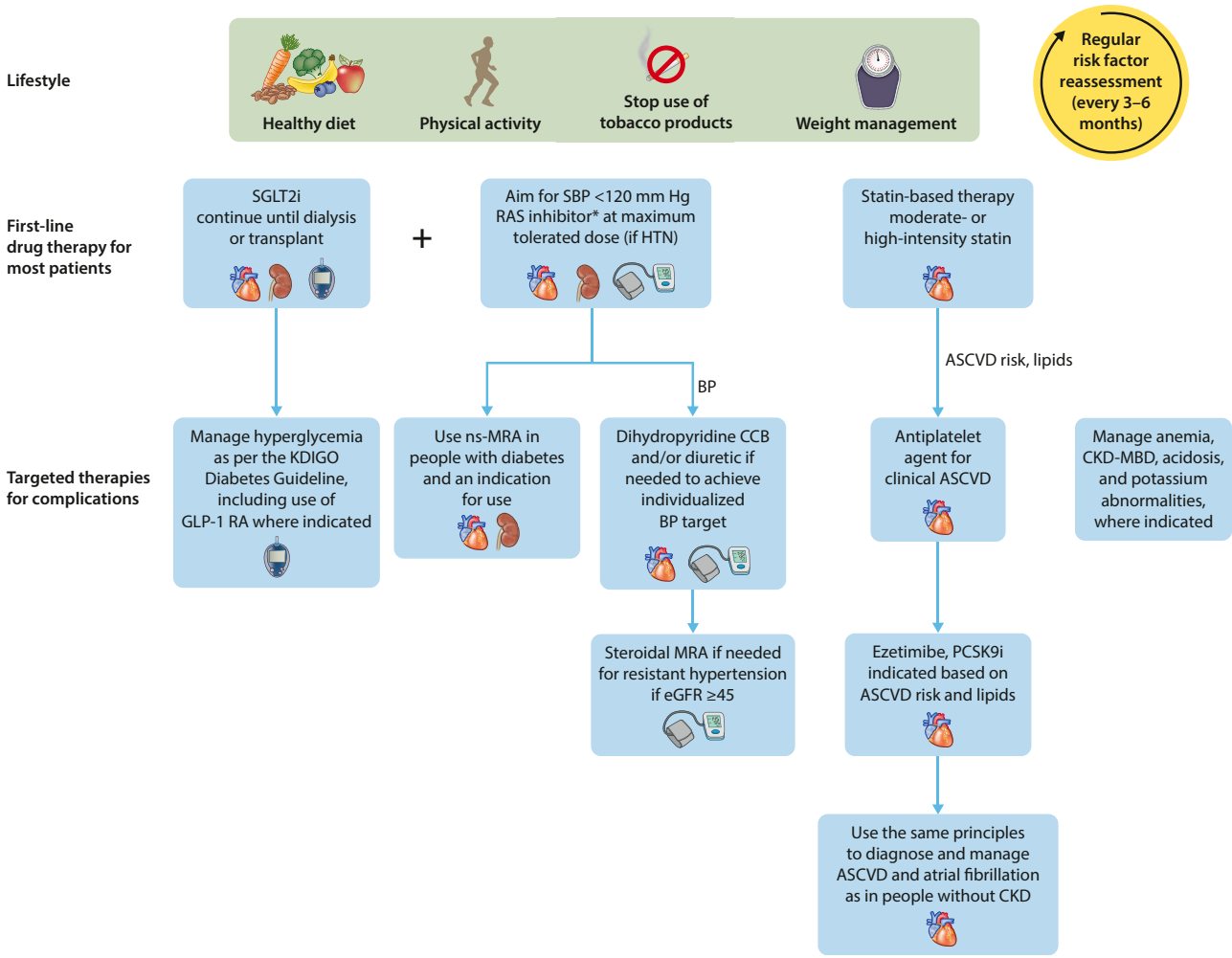

Figure 2 | Holistic approach to chronic kidney disease (CKD) treatment and risk modification. *Angiotensin-converting enzyme inhibitor or angiotensin II receptor blocker should be first-line therapy for blood pressure (BP) control when albuminuria is present, otherwise dihydropyridine calcium channel blocker (CCB) or diuretic can also be considered; all 3 classes are often needed to attain BP targets. Icons presented indicate the following benefits: blood pressure cuff = blood pressure–lowering; glucometer = glucose-lowering; heart = heart protection; kidney = kidney protection; scale = weight management. ASCVD, atherosclerotic cardiovascular disease; CKD-MBD, chronic kidney disease–mineral and bone disorder; eGFR, estimated glomerular filtration rate; GLP-1 RA, glucagon-like peptide-1 receptor agonist; HTN, hypertension; KDIGO, Kidney Disease: Improving Global Outcomes; MRA, mineralocorticoid receptor antagonist; ns-MRA, nonsteroidal mineralocorticoid receptor antagonist; PCSK9i, proprotein convertase subtilisin/kexin type 9 inhibitor; RAS, renin-angiotensin system; SBP, systolic blood pressure; SGLT2i, sodium-glucose cotransporter-2 inhibitor. Modified from Kidney Disease: Improving Global Outcomes Diabetes Work Group. KDIGO 2022 Clinical Practice Guideline for Diabetes Management in Chronic Kidney Disease. *Kidney Int*. 2022;102:S1–S127.[24]

Table 4 | Recommendations and practice points from Chapter 3 of the KDIGO 2024 Clinical Practice Guideline for Evaluation and Management of CKD

Chapter 3. Delaying CKD progression and managing its complications

3.1 CKD treatment and risk modification

Practice Point 3.1.1: Treat people with CKD with a comprehensive treatment strategy to reduce risks of progression of CKD and its associated complications (Figure 17[a]).

3.2 Lifestyle factors

Practice Point 3.2.1: Encourage people with CKD to undertake physical activity compatible with cardiovascular health, tolerance, and level of frailty; achieve an optimal body mass index (BMI); and not to use tobacco products. Referral to providers and programs (e.g., psychologists, renal dietitians or accredited nutrition providers, pharmacists, physical and occupational therapy, and smoking cessation programs) should be offered where indicated and available.

3.2.1 Avoiding use tobacco products

[No specific recommendations or practice points]

3.2.2 Physical activity and optimum weight

Recommendation 3.2.2.1: We recommend that people with CKD be advised to undertake moderate-intensity physical activity for a cumulative duration of at least 150 minutes per week, or to a level compatible with their cardiovascular and physical tolerance *(1D)*.

Practice Point 3.2.2.1: Recommendations for physical activity should consider age, ethnic background, presence of other comorbidities, and access to resources.

Practice Point 3.2.2.2: People with CKD should be advised to avoid sedentary behavior.

Practice Point 3.2.2.3: For people at higher risk of falls, healthcare providers should provide advice on the intensity of physical activity (low, moderate, or vigorous) and the type of exercises (aerobic vs. resistance, or both).

Practice Point 3.2.2.4: Physicians should consider advising/encouraging people with obesity and CKD to lose weight.

Special considerations

Pediatric considerations

Practice Point 3.2.2.5: Encourage children with CKD to undertake physical activity aiming for World Health Organization (WHO)-advised levels (i.e., ≥60 minutes daily) and to achieve a healthy weight.

3.3 Diet

Practice Point 3.3.1: Advise people with CKD to adopt healthy and diverse diets with a higher consumption of plant-based foods compared to animal-based foods and a lower consumption of ultraprocessed foods.

Practice Point 3.3.2: Use renal dietitians or accredited nutrition providers to educate people with CKD about dietary adaptations regarding sodium, phosphorus, potassium, and protein intake, tailored to their individual needs, and severity of CKD and other comorbid conditions.

3.3.1 Protein intake

Recommendation 3.3.1.1: We suggest maintaining a protein intake of 0.8 g/kg body weight/d in adults with CKD G3–G5 *(2C)*.

Practice Point 3.3.1.1: Avoid high protein intake (>1.3 g/kg body weight/d) in adults with CKD at risk of progression.

Practice Point 3.3.1.2: In adults with CKD who are willing and able, and who are at risk of kidney failure, consider prescribing, under close supervision, a very low–protein diet (0.3–0.4 g/kg body weight/d) supplemented with essential amino acids or ketoacid analogs (up to 0.6 g/kg body weight/d).

Practice Point 3.3.1.3: Do not prescribe low- or very low–protein diets in metabolically unstable people with CKD.

Special considerations

Pediatric considerations

Practice Point 3.3.1.4: Do not restrict protein intake in children with CKD due to the risk of growth impairment. The target protein and energy intake in children with CKD G2–G5 should be at the upper end of the normal range for healthy children to promote optimal growth.

Older adults

Practice Point 3.3.1.5: In older adults with underlying conditions such as frailty and sarcopenia, consider higher protein and calorie dietary targets.

3.3.2 Sodium intake

Recommendation 3.3.2.1: We suggest that sodium intake be <2 g of sodium per day (or <90 mmol of sodium per day, or <5 g of sodium chloride per day) in people with CKD *(2C)*.

Practice Point 3.3.2.1: Dietary sodium restriction is usually not appropriate for patients with sodium-wasting nephropathy.

Special considerations

Pediatric considerations

Practice Point 3.3.2.2: Follow age-based Recommended Daily Intake when counseling about sodium intake for children with CKD who have systolic and/or diastolic blood pressure >90th percentile for age, sex, and height.

3.4 Blood pressure control

Recommendation 3.4.1: We suggest that adults with high BP and CKD be treated with a target systolic blood pressure (SBP) of <120 mm Hg, when tolerated, using standardized office BP measurement *(2B)*.

Practice Point 3.4.1: Consider less intensive BP-lowering therapy in people with frailty, high risk of falls and fractures, very limited life expectancy, or symptomatic postural hypotension.

Special considerations

Pediatric considerations

Recommendation 3.4.2: We suggest that in children with CKD, 24-hour mean arterial pressure (MAP) by ambulatory blood pressure monitoring (ABPM) should be lowered to ≤50th percentile for age, sex, and height *(2C)*.

Practice Point 3.4.2: Monitor BP once a year with ABPM and every 3–6 months with standardized auscultatory office BP in children with CKD.

(*Continued on following page*)

Table 4 | (Continued) **Recommendations and practice points from Chapter 3 of the KDIGO 2024 Clinical Practice Guideline for Evaluation and Management of CKD**

Chapter 3. Delaying CKD progression and managing its complications

Practice Point 3.4.3: In children with CKD, when ABPM is not available, it is reasonable to target manual auscultatory office SBP, obtained in a protocol-driven standardized setting, of 50th–75th percentile for age, sex, and height unless achieving this target is limited by signs or symptoms of hypotension.

3.5 Glycemic control

Please refer to the KDIGO 2022 Clinical Practice Guideline for Diabetes Management in Chronic Kidney Disease[24] for specific recommendations, practice points, and research recommendations.

3.6 Renin-angiotensin system inhibitors

Recommendation 3.6.1: We recommend starting renin-angiotensin-system inhibitors (RASi) (angiotensin-converting enzyme inhibitor [ACEi] or angiotensin II receptor blocker [ARB]) for people with CKD and severely increased albuminuria (G1–G4, A3) without diabetes *(1B)*.

Recommendation 3.6.2: We suggest starting RASi (ACEi or ARB) for people with CKD and moderately increased albuminuria (G1–G4, A2) without diabetes *(2C)*.

Recommendation 3.6.3: We recommend starting RASi (ACEi or ARB) for people with CKD and moderately-to-severely increased albuminuria (G1–G4, A2 and A3) with diabetes *(1B)*.

Recommendation 3.6.4: We recommend avoiding any combination of ACEi, ARB, and direct renin inhibitor (DRI) therapy in people with CKD, with or without diabetes *(1B)*.

Practice Point 3.6.1: RASi (ACEi or ARB) should be administered using the highest approved dose that is tolerated to achieve the benefits described because the proven benefits were achieved in trials using these doses.

Practice Point 3.6.2: Changes in BP, serum creatinine, and serum potassium should be checked within 2–4 weeks of initiation or increase in the dose of a RASi, depending on the current GFR and serum potassium.

Practice Point 3.6.3: Hyperkalemia associated with use of RASi can often be managed by measures to reduce the serum potassium levels rather than decreasing the dose or stopping RASi.

Practice Point 3.6.4: Continue ACEi or ARB therapy unless serum creatinine rises by more than 30% within 4 weeks following initiation of treatment or an increase in dose.

Practice Point 3.6.5: Consider reducing the dose or discontinuing ACEi or ARB in the setting of either symptomatic hypotension or uncontrolled hyperkalemia despite medical treatment, or to reduce uremic symptoms while treating kidney failure (estimated glomerular filtration rate [eGFR] <15 ml/min per 1.73 m^2).

Practice Point 3.6.6: Consider starting people with CKD with normal to mildly increased albuminuria (A1) on RASi (ACEi or ARB) for specific indications (e.g., to treat hypertension or heart failure with low ejection fraction).

Practice Point 3.6.7: Continue ACEi or ARB in people with CKD even when the eGFR falls below 30 ml/min per 1.73 m^2.

3.7 Sodium-glucose cotransporter-2 inhibitors (SGLT2i)

Recommendation 3.7.1: We recommend treating patients with type 2 diabetes (T2D), CKD, and an eGFR ≥20 ml/min per 1.73 m^2 with an SGLT2i *(1A)*.

Practice Point 3.7.1: Once an SGLT2i is initiated, it is reasonable to continue an SGLT2i even if the eGFR falls below 20 ml/min per 1.73 m^2, unless it is not tolerated or KRT is initiated.

Practice Point 3.7.2: It is reasonable to withhold SGLT2i during times of prolonged fasting, surgery, or critical medical illness (when people may be at greater risk for ketosis).

Recommendation 3.7.2: We recommend treating adults with CKD with an SGLT2i for the following *(1A)*:

- **eGFR ≥20 ml/min per 1.73 m^2 with urine ACR ≥200 mg/g (≥20 mg/mmol), or**
- **heart failure, irrespective of level of albuminuria.**

Practice Point 3.7.3: SGLT2i initiation or use does not necessitate alteration of frequency of CKD monitoring and the reversible decrease in eGFR on initiation is generally not an indication to discontinue therapy.

Recommendation 3.7.3: We suggest treating adults with eGFR 20 to 45 ml/min per 1.73 m^2 with urine ACR <200 mg/g (<20 mg/mmol) with an SGLT2i *(2B)*.

3.8 Mineralocorticoid receptor antagonists (MRA)

Recommendation 3.8.1: We suggest a nonsteroidal mineralocorticoid receptor antagonist with proven kidney or cardiovascular benefit for adults with T2D, an eGFR >25 ml/min per 1.73 m^2, normal serum potassium concentration, and albuminuria (>30 mg/g [>3 mg/mmol]) despite maximum tolerated dose of RAS inhibitor (RASi) *(2A)*.

Practice Point 3.8.1: Nonsteroidal MRA are most appropriate for adults with T2D who are at high risk of CKD progression and cardiovascular events, as demonstrated by persistent albuminuria despite other standard-of-care therapies.

Practice Point 3.8.2: A nonsteroidal MRA may be added to a RASi and an SGLT2i for treatment of T2D and CKD in adults.

Practice Point 3.8.3: To mitigate risk of hyperkalemia, select people with consistently normal serum potassium concentration and monitor serum potassium regularly after initiation of a nonsteroidal MRA (Figure 26[b]).

Practice Point 3.8.4: The choice of a nonsteroidal MRA should prioritize agents with documented kidney or cardiovascular benefits.

Practice Point 3.8.5: A steroidal MRA may be used for treatment of heart failure, hyperaldosteronism, or refractory hypertension, but may cause hyperkalemia or a reversible decline in glomerular filtration, particularly among people with a low GFR.

3.9 Glucagon-like peptide-1 receptor agonists (GLP-1 RA)

Recommendation 3.9.1: In adults with T2D and CKD who have not achieved individualized glycemic targets despite use of metformin and SGLT2 inhibitor treatment, or who are unable to use those medications, we recommend a long-acting GLP-1 RA *(1B)*.

Practice Point 3.9.1: The choice of GLP-1 RA should prioritize agents with documented cardiovascular benefits.

(*Continued on following page*)

Table 4 | (Continued)

Chapter 3. Delaying CKD progression and managing its complications

3.10 Metabolic acidosis

Practice Point 3.10.1: In people with CKD, consider use of pharmacological treatment with or without dietary intervention to prevent development of acidosis with potential clinical implications (e.g., serum bicarbonate <18 mmol/l in adults).

Practice Point 3.10.2: Monitor treatment for metabolic acidosis to ensure it does not result in serum bicarbonate concentrations exceeding the upper limit of normal and does not adversely affect BP control, serum potassium, or fluid status.

3.11 Hyperkalemia in CKD

3.11.1 Awareness of factors impacting on potassium measurement

Practice Point 3.11.1.1: Be aware of the variability of potassium laboratory measurements as well as factors and mechanisms that may influence potassium measurement including diurnal and seasonal variation, plasma versus serum samples, and the actions of medications.

3.11.2 Potassium exchange agents

Practice Point 3.11.2.1: Be aware of local availability or formulary restrictions with regard to the pharmacologic management of nonemergent hyperkalemia.

3.11.3 Timing to recheck potassium after identifying moderate and severe hyperkalemia in adults

[No specific recommendations or practice points]

3.11.4 Managing hyperkalemia

[No specific recommendations or practice points]

3.11.5 Dietary considerations

Practice Point 3.11.5.1: Implement an individualized approach in people with CKD G3–G5 and emergent hyperkalemia that includes dietary and pharmacologic interventions and takes into consideration associated comorbidities and quality of life (QoL). Assessment and education through a renal dietitian or an accredited nutrition provider are advised.

Practice Point 3.11.5.2: Provide advice to limit the intake of foods rich in bioavailable potassium (e.g., processed foods) for people with CKD G3–G5 who have a history of hyperkalemia or as a prevention strategy during disease periods in which hyperkalemia risk may be a concern.

3.12 Anemia

Please refer to the *KDIGO Clinical Practice Guideline for Anemia in Chronic Kidney Disease* publications for specific recommendations, selection, and dosing of specific therapeutic agents, and research recommendations.

3.13 CKD-Mineral Bone Disorder (CKD-MBD)

Please refer to the *KDIGO 2017 Clinical Practice Guideline Update for the Diagnosis, Evaluation, Prevention, and Treatment of Chronic Kidney Disease–Mineral and Bone Disorder (CKD-MBD)* for specific recommendations, selection, dosing of specific therapeutic agents, and research recommendations.

3.14 Hyperuricemia

Recommendation 3.14.1: We recommend people with CKD and symptomatic hyperuricemia should be offered uric acid–lowering intervention (*1C*).

Practice Point 3.14.1: Consider initiating uric acid–lowering therapy for people with CKD after their first episode of gout (particularly where there is no avoidable precipitant or serum uric acid concentration is >9 mg/dl [535 μmol/l]).

Practice Point 3.14.2: Prescribe xanthine oxidase inhibitors in preference to uricosuric agents in people with CKD and symptomatic hyperuricemia.

Practice Point 3.14.3: For symptomatic treatment of acute gout in CKD, low-dose colchicine or intra-articular/oral glucocorticoids are preferable to nonsteroidal anti-inflammatory drugs (NSAIDs).

Practice Point 3.14.4: Nonpharmacological interventions which may help prevent gout include limiting alcohol, meats, and high-fructose corn syrup intake.

Recommendation 3.14.2: We suggest not using agents to lower serum uric acid in people with CKD and asymptomatic hyperuricemia to delay CKD progression (*2D*).

3.15 Cardiovascular disease (CVD) and additional specific interventions to modify risk

3.15.1 Lipid management

Recommendation 3.15.1.1: In adults aged ≥50 years with eGFR <60 ml/min per 1.73 m^2 but not treated with chronic dialysis or kidney transplantation (GFR categories G3a–G5), we recommend treatment with a statin or statin/ezetimibe combination (*1A*).

Recommendation 3.15.1.2: In adults aged ≥50 years with CKD and eGFR ≥60 ml/min per 1.73 m^2 (GFR categories G1–G2), we recommend treatment with a statin (*1B*).

Recommendation 3.15.1.3: In adults aged 18–49 years with CKD but not treated with chronic dialysis or kidney transplantation, we suggest statin treatment in people with one or more of the following (*2A*):

- **known coronary disease (myocardial infarction or coronary revascularization),**
- **diabetes mellitus,**
- **prior ischemic stroke, or**
- **estimated 10-year incidence of coronary death or nonfatal myocardial infarction >10%.**

Practice Point 3.15.1.1: Estimate 10-year cardiovascular risk using a validated risk tool.

Practice Point 3.15.1.2: In people with CKD, choose statin-based regimens to maximize the absolute reduction in low-density lipoprotein (LDL) cholesterol to achieve the largest treatment benefits.

(*Continued on following page*)

Table 4 | (Continued) **Recommendations and practice points from Chapter 3 of the KDIGO 2024 Clinical Practice Guideline for Evaluation and Management of CKD**

Chapter 3. Delaying CKD progression and managing its complications
Practice Point 3.15.1.3: In adults with CKD aged 18–49, a lower (i.e., <10%) estimated 10-year incidence of coronary death or nonfatal myocardial infarction may also be appropriate thresholds for initiation of statin-based therapy.
Practice Point 3.15.1.4: Consider prescribing proprotein convertase subtilisin/kexin type 9 (PCSK-9) inhibitors to people with CKD who have an indication for their use.
Practice Point 3.15.1.5: Consider a plant-based "Mediterranean-style" diet in addition to lipid-modifying therapy to reduce cardiovascular risk.
3.15.2 Use of antiplatelet therapy
Recommendation 3.15.2.1: We recommend oral low-dose aspirin for prevention of recurrent ischemic cardiovascular disease events (i.e., secondary prevention) in people with CKD and established ischemic cardiovascular disease (*1C*).
Practice Point 3.15.2.1: Consider other antiplatelet therapy (e.g., $P2Y_{12}$ inhibitors) when there is aspirin intolerance.
3.15.3 Invasive versus intensive medical therapy for coronary artery disease
Recommendation 3.15.3.1: We suggest that in stable stress-test confirmed ischemic heart disease, an initial conservative approach using intensive medical therapy is an appropriate alternative to an initial invasive strategy (*2D*).
Practice Point 3.15.3.1: Initial management with an invasive strategy may still be preferable for people with CKD with acute or unstable coronary disease, unacceptable levels of angina (e.g., patient dissatisfaction), left ventricular systolic dysfunction attributable to ischemia, or left main disease.
3.16 CKD and atrial fibrillation
Practice Point 3.16.1: Follow established strategies for the diagnosis and management of atrial fibrillation (Figure 40[c]).
Recommendation 3.16.1: We recommend use of non–vitamin K antagonist oral anticoagulants (NOACs) in preference to vitamin K antagonists (e.g., warfarin) for thromboprophylaxis in atrial fibrillation in people with CKD G1–G4 (*1C*).
Practice Point 3.16.2: NOAC dose adjustment for GFR is required, with caution needed at CKD G4–G5.
Practice Point 3.16.3: Duration of NOAC discontinuation before elective procedures needs to consider procedural bleeding risk, NOAC prescribed, and level of GFR (Figure 44[d]).

CKD, chronic kidney disease; KDIGO, Kidney Disease: Improving Global Outcomes.
[a]Figure 17 in full guideline.
[b]Figure 26 in full guideline.
[c]Figure 40 in full guideline.
[d]Figure 44 in full guideline.

plans based on the best evidence for different people at different points in their kidney journey. Salt intake and blood pressure management are concordant with other guidance documents, and again caveats for targets in vulnerable or frail populations are highlighted. Measurements using 24-hour ambulatory devices are underscored, as is the interpretation of those readouts for children.

Recommendations for using renin-angiotensin system inhibitors, where tolerated, in those with and without diabetes and in those with moderately high urine albumin excretion are clearly articulated, as is the value of these medications in the context of heart failure and CKD. Guidance for reduction or cessation of medication for those intolerant (due to side effects or hypotension) is offered, as are reassurances as to when to maintain people on these agents despite changes in bloodwork (serum creatinine, potassium, etc.).

There is an abundance of literature on which to base guidance on the use of SGLT2 inhibitors to both delay CKD progression and reduce cardiovascular complications. Nuances for those with and without diabetes, heart failure, and for those with higher and lower urine ACR levels are clearly articulated. Guidance for the use of mineralocorticoid antagonists (both steroidal and nonsteroidal) is also offered, given their value in blood pressure management, heart failure treatment, and delay of progression of CKD in those with and without diabetes.

The treatment of metabolic acidosis to delay progression of CKD has not been proven; however, it is recognized that acidosis can lead to exacerbation of nutritional issues (anorexia and protein wasting), bone disease, and other consequences. These can be addressed with both dietary interventions and medications. Suggestions to evaluate the risk-benefit ratio and goals of therapy are described, and the threshold for aggressive intervention has been moved to <18 mmol/l from <22 mmol/l in the previous KDIGO guideline.

Hyperkalemia is discussed in terms of frequency of occurrence, "expected" values for potassium at different levels of GFR, and treatment strategies, which highlight newer attitudes toward dietary advice (avoidance of highly processed foods, but not of fruits and vegetables), an improved understanding of different factors that impact potassium, and targeted therapy based on thorough assessment and context.

Newer data on the value of treating hyperuricemia were evaluated. No data were found to support targeting hyperuricemia in the absence of symptoms (e.g., gout or tophi formation).

The treatment of common laboratory abnormalities of hemoglobin, parathyroid hormone, and phosphate follows current KDIGO guideline documents on these specific entities. Importantly, tables are provided describing "expected" values for common laboratory tests by eGFR so that clinicians may be able to better identify aberrant values that require additional work-up or review.

A section on cardiovascular risk factor modification for both atherosclerotic disease (myocardial infarction, stroke, and peripheral vascular disease) and atrial fibrillation is offered whereby specific interventions are highlighted and calculation of risk scores promoted to guide initiation or modification of

therapy. The use of statins, antiplatelet therapy, and invasive versus intensive medical therapy is addressed in detail, citing and interpreting an extensive evidence base.

The chapter emphasizes the need to evaluate people with CKD comprehensively in order to address both progressive CKD and its attendant comorbidities in an integrated manner and draws on literature to support this holistic approach (Figure 2[24]).

Chapter 4: Medical management and drug stewardship in CKD

This chapter reminds HCPs and people with CKD about the importance of assessing medications in the context of GFR, and given that GFR changes over time, it emphasizes the need for and value of regular reassessment (Table 5). Drugs with narrow therapeutic windows need to be dosed according to the most accurate assessment of GFR, which may require direct measurement.

Understanding of drug metabolism and excretion is poor in specific patient groups (children and in persons of childbearing age), and the impact of concomitant hormonal therapy (for any reason, in male or female individuals) is not known. Thus, we should proceed with caution and ensure that we gather more data in these areas to better inform care for diverse individuals across the life spectrum.

Chapter 4 also promotes the concepts of recognizing the risks of polypharmacy, the potential value of deprescribing, and the need for good drug stewardship, both for people with CKD as well as providers. Accessible education and communication for people with CKD will support efforts at health literacy but also potentially mitigate issues of nonadherence. Given the very large number of drugs that people with CKD are prescribed, this is an important component of care, ensuring that people are prescribed the right drugs for the right reasons at the right dose.

Lastly, the chapter highlights new literature that suggests that intravenous contrast does not carry large risks in people with CKD and that imaging studies should be performed based on information they provide and how and if they will change management. The true low risk of associated acute kidney injury is highlighted and will require ongoing education of all HCPs and people with CKD to enable appropriate timely diagnostic imaging.

Chapter 5: Optimal models of care

This chapter addresses the value of referral to nephrology specialists for comprehensive evaluation and work-up and the importance of access to multidisciplinary care teams, while recognizing the associated cost(s) and the nonavailability of multidisciplinary care teams in different regions (Table 5). There is an emphasis on standardized regular assessment of symptoms, and the use of validated questionnaires to help both people with CKD and HCPs appreciate changes over time and the impact of symptom management (Figure 3).

The need for planned and extended transitions from pediatric to adult care is highlighted, as is the importance of true team-based care models. The value of supportive care and comprehensive conservative management is described for those who choose not to or who cannot access kidney replacement therapy. The use of technology, digital platforms, and tools, including, but not limited to, virtual care, to help teams and individuals access the totality of the value of the team and resources is described. We recognize that these

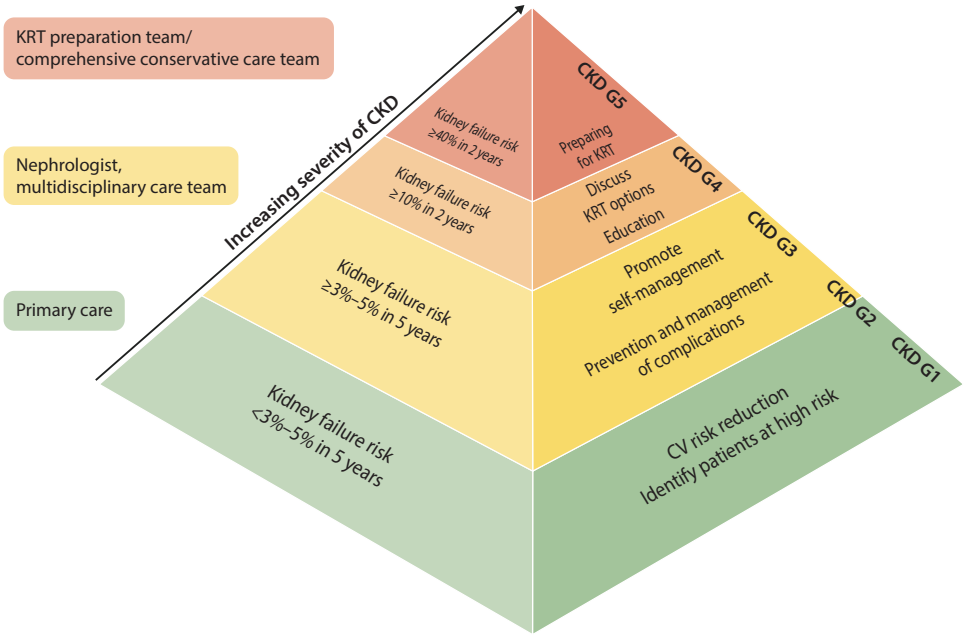

Figure 3 | Optimal care model by increasing severity of chronic kidney disease (CKD). CV, cardiovascular; KRT, kidney replacement therapy.

Table 5 | Recommendations and practice points from Chapters 4 and 5 of the KDIGO 2024 Clinical Practice Guideline for Evaluation and Management of CKD

Chapter 4. Medication management and drug stewardship in CKD

4.1 Medication choices and monitoring for safety

Practice Point 4.1.1: People with CKD may be more susceptible to the nephrotoxic effects of medications. When prescribing such medications to people with CKD, always consider the benefits versus potential harms.

Practice Point 4.1.2: Monitor eGFR, electrolytes, and therapeutic medication levels, when indicated, in people with CKD receiving medications with narrow therapeutic windows, potential adverse effects, or nephrotoxicity, both in outpatient practice and in hospital settings.

Practice Point 4.1.3: Review and limit the use of over-the-counter medicines and dietary or herbal remedies that may be harmful for people with CKD.

Practice Point 4.1.4: When prescribing medications to people with CKD who are of child-bearing potential, always review teratogenicity potential and provide regular reproductive and contraceptive counseling in accordance with the values and preferences of the person with CKD.

4.2 Dose adjustments by level of GFR

Practice Point 4.2.1: Consider GFR when dosing medications cleared by the kidneys.

Practice Point 4.2.2: For most people and clinical settings, validated eGFR equations using SCr are appropriate for drug dosing.

Practice Point 4.2.3: Where more accuracy is required for drug-related decision-making (e.g., dosing due to narrow therapeutic or toxic range), drug toxicity, or clinical situations where eGFRcr estimates may be unreliable, use of equations that combine both creatinine and cystatin C, or measured GFR may be indicated.

Practice Point 4.2.4: In people with extremes of body weight, eGFR nonindexed for body surface area (BSA) may be indicated, especially for medications with a narrow therapeutic range or requiring a minimum concentration to be effective.

Practice Point 4.2.5: Consider and adapt drug dosing in people where GFR, non-GFR determinants of the filtration markers, or volume of distribution are not in a steady state.

4.3 Polypharmacy and drug stewardship

Practice Point 4.3.1: Perform thorough medication review periodically and at transitions of care to assess adherence, continued indication, and potential drug interactions because people with CKD often have complex medication regimens and are seen by multiple specialists.

Practice Point 4.3.2: If medications are discontinued during an acute illness, communicate a clear plan of when to restart the discontinued medications to the affected person and healthcare providers, and ensure documentation in the medical record.

Practice Point 4.3.3: Consider planned discontinuation of medications (such as metformin, ACEi, ARBs, and SGLT2i) in the 48–72 hours prior to elective surgery or during the acute management of adverse effects as a precautionary measure to prevent complications. However, note that failure to restart these medications after the event or procedure may lead to unintentional harm (see Practice Point 4.3.2).

4.3.1 Strategies to promote drug stewardship

Practice Point 4.3.1.1: Educate and inform people with CKD regarding the expected benefits and possible risks of medications so that they can identify and report adverse events that can be managed.

Practice Point 4.3.1.2: Establish collaborative relationships with other healthcare providers and pharmacists and/or use tools to ensure and improve drug stewardship in people with CKD to enhance management of their complex medication regimens.

4.4 Imaging studies

Practice Point 4.4.1: Consider the indication for imaging studies in accordance with general population indications. Risks and benefits of imaging studies should be determined on an individual basis in the context of their CKD.

4.4.1 Radiocontrast: intra-arterial and intravenous dye studies

Practice Point 4.4.1.1: Assess the risk for AKI in people with CKD receiving intra-arterial contrast for cardiac procedures using validated tools.

Practice Point 4.4.1.2: The intravenous administration of radiocontrast media can be managed in accordance with consensus statements from the radiology societies in people with AKI or GFR <60 ml/min per 1.73 m^2 (CKD G3a–G5) undergoing elective investigation.

4.4.2 Gadolinium-containing contrast media

Practice Point 4.4.2.1: For people with GFR <30 ml/min per 1.73 m^2 (CKD G4–G5) who require gadolinium-containing contrast media, preferentially offer them American Colleague of Radiology group II and III gadolinium-based contrast agents.

Chapter 5. Optimal models of care

5.1 Referral to specialist kidney care services

Practice Point 5.1.1: Refer adults with CKD to specialist kidney care services in the circumstances listed in Figure 48.[a]

Special considerations

Pediatric considerations

Practice Point 5.1.2: Refer children and adolescents to specialist kidney care services in the following circumstances:

- an ACR of 30 mg/g (3 mg/mmol) OR a PCR of 200 mg/g (20 mg/mmol) or more, confirmed on a repeat first morning void sample, when well and not during menstruation,
- persistent hematuria,
- any sustained decrease in eGFR,
- hypertension,
- kidney outflow obstruction or anomalies of the kidney and urinary tract,
- known or suspected CKD, or
- recurrent urinary tract infection.

5.2 Symptoms in CKD

5.2.1 Prevalence and severity of symptoms

[No specific recommendations or practice points]

(*Continued on following page*)

Table 5 | (Continued)

Chapter 5. Optimal models of care

5.2.2 Identification and assessment of symptoms

Practice Point 5.2.2.1: Ask people with progressive CKD about uremic symptoms (e.g., reduced appetite, nausea, and level of fatigue/lethargy) at each consultation using a standardized validated assessment of uremic symptoms tool.

5.2.3 Management of common symptoms for people with CKD

Practice Point 5.2.3.1: Use evidence-informed management strategies to support people to live well with CKD and improve their health-related quality of life.

Practice Point 5.2.3.2: Screen people with CKD G4–G5, aged >65, poor growth (pediatrics), or symptoms such as involuntary weight loss, frailty, or poor appetite twice annually for malnutrition using a validated assessment tool.

Practice Point 5.2.3.3: Enable availability of appropriate medical nutrition therapy for people with signs of malnutrition, ideally under the supervision of renal dietitians or accredited nutrition providers if not available.

5.3 Team-based integrated care

Practice Point 5.3.1: Enable access to a patient-centered multidisciplinary care team consisting of dietary counseling, medication management, education, and counseling about different KRT modalities, transplant options, dialysis access surgery, and ethical, psychological, and social care for people with CKD.

Practice Point 5.3.2: Education programs that also involve care partners where indicated are important to promote informed, activated people with CKD.

Practice Point 5.3.3: Consider the use of telehealth technologies including web-based, mobile applications, virtual visiting, and wearable devices in the delivery of education and care.

Special considerations

Pediatric considerations

5.3.1 Transition from pediatric to adult care

5.3.1.1 Pediatric providers

Practice Point 5.3.1.1.1: Prepare adolescents and their families for transfer to adult-oriented care starting at 11–14 years of age by using checklists to assess readiness and guide preparation, and by conducting part of each visit without the parent/guardian present (Figure 55[b]).

Practice Point 5.3.1.1.2: Provide a comprehensive written transfer summary, and ideally an oral handover, to the receiving healthcare providers including all relevant medical information as well as information about the young person's cognitive abilities and social support (Figure 55[b]).

Practice Point 5.3.1.1.3: Transfer young people to adult care during times of medical and social stability where possible.

5.3.1.2 Adult providers

Practice Point 5.3.1.2.1: Recognize that young people under 25 years of age with CKD are a unique population at high risk for adverse outcomes at least in part due to physiologic incomplete brain maturation.

Practice Point 5.3.1.2.2: Encourage young people to informally visit the adult care clinic to which they will be transferred before the first appointment (Figure 55[b]).

Practice Point 5.3.1.2.3: Assess young people with CKD more frequently than older people with the same stage of CKD and, with the agreement of the young person, include the caregivers or significant other of the young person in their care, at least in the first 1–3 years following transfer from pediatric care (Figure 55[b]).

5.4 Timing the initiation of dialysis

Practice Point 5.4.1: Initiate dialysis based on a composite assessment of a person's symptoms, signs, QoL, preferences, level of GFR, and laboratory abnormalities.

Practice Point 5.4.2: Initiate dialysis if the presence of one or more of the following situations is evident (Table 41[c]). This often but not invariably occurs in the GFR range between 5 and 10 ml/min per 1.73 m^2.

Practice Point 5.4.3: Consider planning for preemptive kidney transplantation and/or dialysis access in adults when the GFR is <15–20 ml/min per 1.73 m^2 or risk of KRT is >40% over 2 years.

Special considerations

Pediatric considerations

Practice Point 5.4.4: In children, in addition to the adult indications for dialysis, poor growth refractory to optimized nutrition, growth hormone, and medical management is an indication for initiating KRT.

Practice Point 5.4.5: Pursue living or deceased donor preemptive kidney transplantation as the treatment of choice for children in whom there is evidence of progressive and irreversible CKD. The eGFR at which preemptive transplantation should be undertaken will depend on multiple factors including the age and size of the child and the rate of progression of kidney failure but will usually be between eGFR 5–15 ml/min per 1.73 m^2.

5.5 Structure and process of supportive care and comprehensive conservative management

Practice Point 5.5.1: Inform people with CKD about the options for KRT and comprehensive conservative care.

Practice Point 5.5.2: Support comprehensive conservative management as an option for people who choose not to pursue KRT.

Practice Point 5.5.3: Provide access to resources that enable the delivery of advanced care planning for people with a recognized need for end-of-life care, including those people undergoing comprehensive conservative care.

CKD, chronic kidney disease; KDIGO, Kidney Disease: Improving Global Outcomes; QoL, quality of life.

[a]Figure 48 in full guideline.

[b]Figure 55 in full guideline.

[c]Table 41 in full guideline.

technologies are evolving rapidly so that definitive evaluation of the value, harms, and benefits of these is not known.

There is an emphasis on advanced care planning, directed specifically to those choosing supportive care, but also recognizing that all people with chronic diseases do need to ensure that plans addressing future healthcare states are known to all.

Conclusion

Research recommendations are offered in the last chapter in some detail to signpost priority areas for the field, such as improving diagnostic and dynamic tests of kidney health and disease, improving and evaluating the implementation of validated prediction equations in specific situations, and testing different combinations of disease-modifying drugs with dietary regimens, to name but a few.

This comprehensive guidance document based on current best evidence indicates some exciting new approaches to management strategies and treatment options for people living with CKD, with the goal of improving symptom management, disease modification, and offering person-centered approaches, while also recognizing the heterogeneity of CKD. The fact that new therapies exist and others are being evaluated heralds an exciting time for people living with kidney diseases, their families, and the HCP.

DISCLOSURE

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