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CLINICAL GUIDELINE

Evaluation and Management of Chronic Kidney Disease: Synopsis of the Kidney Disease: Improving Global Outcomes 2024 Clinical Practice Guideline

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Description: The Kidney Disease: Improving Global Outcomes (KDIGO) organization updated its existing clinical practice guideline in 2024 to provide guidance on the evaluation, management, and treatment of chronic kidney disease (CKD) in adults and children who are not receiving kidney replacement therapy.

Methods: The KDIGO CKD Guideline Work Group defined the scope of the guideline and determined topics for systematic review. An independent Evidence Review Team systematically reviewed the evidence and graded the certainty of evidence for each of the review topics. Latest searches of the English-language literature were done in July 2023. Final modification of the guideline was informed by a public review process during summer of 2023 involving registered stakeholders.

Recommendations: The full guideline included 28 recommendations and 141 practice points. This synopsis focuses on the recommendations that have the greatest evidence. Practice points reflect the expert opinion of the group where evidence is not that strong. Recommendations include greater emphasis on cystatin C for assessment of glomerular filtration rate, point-of-care testing in remote areas, a shift to an individualized risk-based approach to predict kidney failure, sodium–glucose cotransporter-2 inhibitors for some patients with CKD with and without diabetes, and statin use for adults older than 50 years and CKD. Together the recommendations and practice points provide guidance for how to evaluate and manage persons with CKD.

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The 2024 update of the Kidney Disease: Improving Global Outcomes (KDIGO) chronic kidney disease (CKD) guideline applies to all persons with CKD not receiving kidney replacement therapy (KRT) (1). The updated guideline describes the entire patient pathway from early diagnosis to decisions about KRT method, including conservative care. This synopsis does not reflect the entire guideline, but highlights level 1 or 2 recommendations and ungraded practice points that have evolved since 2012 (2). Practice points represent consensus-based expert judgment of the Work Group (WG) and are intended to aid implementation of a recommendation or guide practice where evidence generation is considered impossible or absurd. Together the recommendations and practice points provide guidance for how to evaluate and manage persons with CKD.

The guideline emphasizes the importance of tailored care, which varies over the life course from infants to old age. Different approaches and prioritization depend on specific aspects of individual situations. There are sex-dependent variations in genetics, physiology, immunology, and anatomy as well as gender-based factors (for example, identity, roles, and relations) that influence CKD pathophysiology, progression, and responses to therapy.

Many of the diagnostics, therapies, and strategies recommended by the guideline will only be feasible in higher-resource settings. The WG has integrated perspectives from representatives of low-, middle-, and high-resource countries, acknowledging the limitations in access to care in some regions. The guideline raises awareness about global inequities and highlights the evidence base supporting best care, thus facilitating better kidney health for all.

METHODS

Full methods for the guideline development process are described in the "KDIGO 2024 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease" (1). The guideline follows international

See also:

Web-Only
Supplement

standards for guideline development (3, 4) and has been reported in accordance with the AGREE II (Appraisal of Guidelines for Research and Evaluation II) reporting checklist (5). The WG comprised pediatric, adult, and geriatric nephrologists, including both dialysis and transplant specialists; primary care physicians; internal medicine physicians; clinical scientists; dietitians; nurses; women's health experts; clinical trialists; epidemiologists; experts in medical decision making and public health; as well as persons living with CKD. Conflicts of interest were fully disclosed and managed in accordance with the KDIGO Methods Manual (6). The conflicts of interest for each WG member have been published alongside the guideline. Johns Hopkins University, the Evidence Review Team, conducted literature searches for each topic through July 2023. Evidence synthesis and meta-analysis were done if there were 2 or more studies that were sufficiently similar with respect to key variables (population characteristics, study duration, and comparisons). Interventions in the same class were combined when reporting outcomes. The findings of the evidence reviews were summarized into tables using standard Cochrane and GRADE (Grading of Recommendations Assessment, Development and Evaluation) methods (2, 7, 8). Recommendations were graded as either level 1 ("we recommend") or level 2 ("we suggest"); the strength of a recommendation was based on a judgment by the WG using the GRADE Evidence-to-Decision framework, which considers the balance of benefits and harms, the certainty of the evidence, perceived patient values and preferences, and resource implications (**Supplement Table 1**, available at Annals.org). The certainty of evidence was graded as high (A), moderate (B), low (C), or very low (D) (**Supplement Table 2**, available at Annals.org). The intended use of level 1 and level 2 recommendations is summarized in **Supplement Table 3** (available at Annals.org) (9). In addition to the 28 graded recommendations, 141 ungraded practice points were developed to provide clinicians with expert input or guidance for implementation.

Evaluation of CKD

CKD Staging System and Testing of CKD With Renewed Emphasis on Urine Albumin-to-Creatinine Ratio

We reinforce the CGA (Cause, GFR, ACR) classification concept and the use of the 2 domains, glomerular filtration rate (GFR) and urine albumin-to-creatinine ratio (uACR), for staging individual risk prediction and in the context of newly available drugs. We reemphasize the importance of uACR testing and monitoring in addition to GFR, recognizing that this simple test is evaluated only in a third of population studies worldwide (10). We encourage testing persons at risk for (for example, persons with hypertension, diabetes, multisystem diseases, or exposure to potentially nephrotoxic drugs) and with CKD using both uACR and GFR (Practice Point 1.1.1.1, **Table 1**) (11).

Use of Cystatin C in GFR Assessment and Understanding Limitations in GFR Assessment Tools

In adults at risk for CKD, we recommend using creatinine-based estimated GFR (eGFRcr). If cystatin C is available, the GFR category should be estimated from the combination of creatinine and cystatin C (eGFRcr-cys) (1B, Recommendation 1.1.2.1, **Table 1**) (12–32). The evidence for this recommendation is that compared with equations based on creatinine and cystatin C alone, equations using both creatinine and cystatin C come closest to the gold standard (measured GFR) most consistently demonstrating higher accuracy (**Supplement Table 4**, available at Annals.org), and data demonstrate that the combined eGFRcr-cys equation is superior for distinguishing GFR risk stages compared with eGFRcr (12–32). The guideline aims to improve clinician understanding of patient characteristics (biological factors) that influence creatinine or cystatin C impacting eGFR results (33–37). By understanding these limitations, clinicians may choose the most appropriate biomarker for an individual, which upholds our goal toward more personalized approaches. Clinically significant biological factors influencing creatinine include extremes of muscle mass, malnutrition, dietary intake, and drugs that impair tubular secretion of creatinine. Factors potentially affecting cystatin C are smoking, chronic inflammation, adiposity, cancer, chemotherapy, thyroid function, and glucocorticoid excess (**Table 2**). Some conditions may influence both biomarkers. Thus, we recommend using eGFRcr-cys in clinical situations when eGFRcr is less accurate and GFR affects clinical decision making (1C, Recommendation 1.2.2.1, **Table 1**; **Table 2**) because equations using the combination of both markers may mitigate the other, allowing a "truer" estimate of GFR (12, 15, 18–22, 25, 26, 30, 31, 48). Again, this recommendation is based on studies demonstrating better accuracy of an equation based on both markers compared with just one. In 2 large-scale studies of general population cohorts and clinical populations in North America and Europe, the P_{30} (defined as the percentage of the eGFR values within ±30% of measured GFR) using eGFRcr-cys was in the range of 90% (30, 49). This observation was confirmed for other populations and countries (Brazil, Congo, Pakistan, Singapore, Japan, and China) (16, 17, 22, 27, 32, 34, 50). For those who require a precise and definitive GFR, we encourage measured GFR using exogenous markers, which are insensitive to such biological factors.

Point-of-Care Testing

To highlight the value of early detection, 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, Recommendation 1.4.1, **Table 1**) (38–40). The recommendation for point-of-care creatinine is based on a systematic review of 54 studies on eGFR and serum creatinine diagnostic accuracy as well as correlation and bias of point-of-care creatinine tests compared with laboratory-based tests plus an additional study in a pediatric population in Uganda overall covering

Table 1. Key Statements From the KDIGO 2024 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease

Evaluation 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).

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).

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 2**) (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 (**Supplement Table 4**).

Practice Point 1.2.4.1: Use the same equation within geographical regions (as defined locally [e.g., continent, country, 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.

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).

Risk assessment 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 2.3.1: 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.

Delaying CKD progression and managing its associated complications

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).

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).

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

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).

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%.

ACR = albumin-to-creatinine ratio; KDIGO = Kidney Disease: Improving Global Outcomes; SGLT2i = sodium–glucose cotransporter-2 inhibitors.
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Table 2. Indications for Use of Cystatin C (or Creatinine) for Assessment of GFR

Domain	Specific Clinical Condition	Cause of Decreased Accuracy of Creatinine (and/or Cystatin C)	Comments on GFR Evaluation
Body habitus and changes in muscle mass	Eating disorders (35)	Biological factors of SCr	eGFRcys may be appropriate if no comorbid illness other than reduction in muscle mass. Suggest eGFRcr-cys in those with comorbid illness.
	Extreme sport/exercise/ body builder	Biological factors of SCr	eGFRcys may be appropriate if an increase in muscle mass is the only abnormality.
	Above-knee amputation (36)	Biological factors 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	Biological factors of SCr	eGFRcys may be appropriate in those without other comorbid illness. Suggest eGFRcr-cys in those with comorbid illness.
	Class III obesity*†	Biological factors of SCr and SCys	eGFRcr-cys demonstrated to be most accurate.
Lifestyle	Smoking (37–39)	Biological factors of SCys	Minimal data, suggest eGFRcr if no changes to biologic factors of SCr or comorbid illness.
Diet	Low-protein diet	Biological factors of SCr	Minimal data, suggest eGFRcys may be appropriate if no changes to biologic factors of SCys or comorbid illness.
	Keto diets	Biological factors of SCr	
	Vegetarian	Biological factors of SCr	
	High-protein diets and creatine supplements	Biological factors of SCr	
Illness other than CKD	Malnutrition	Chronic illness, presumed effect on biological factors of SCr and SCys	eGFRcr-cys because of coexistence of malnutrition and inflammation Suggest using mGFR for treatment decisions based on the level of GFR.
	Cancer* (34, 40–43)	Chronic illness, presumed effect on biological factors 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 (44, 45)	Chronic illness, presumed impact on biological factors 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* (18, 46, 47)	Chronic illness, presumed impact on biological factors 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‡	Chronic illness, presumed impact on biological factors 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 (33)	Chronic illness, presumed impact on biological factors 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)	Biological factors of SCr. Effect on SCys not known	Physiological effect on SCys unknown, suggest eGFRcr-cys.
	Decreases in tubular secretion	Biological factors 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	Biological factors 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.

CKD = chronic kidney disease; eGFR = estimated glomerular filtration rate; eGFRcr = creatinine-based eGFR; eGFRcys = cystatin C–based eGFR; eGFRcr-cys = creatinine and cystatin C–based eGFR; GFR = glomerular filtration rate; mGFR = measured glomerular filtration rate; SCr = serum creatinine; SCys = serum cystatin C.

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* 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. doi:10.1016/j.xkme.2023.100727

† Obesity class III definition varies by region but commonly body mass index >40 or >35 kg/m^2.

‡ Catabolic consuming disease may include tuberculosis, AIDS, hematologic malignancies, and severe skin diseases. There are no data with mGFR to evaluate this directly.

3 devices that all demonstrated acceptable accuracy at lower levels of eGFR (39, 40). For albumin, another systematic review evaluated the diagnostic accuracy of quantitative and semiquantitative protein or albumin urine dipstick tests compared with laboratory tests (38). The certainty of evidence was rated as low for point-of-care creatinine and very low for albumin. These tests may be less accurate than laboratory testing (leading to misdiagnosis or misclassification) and do need calibration to mitigate potential measurement errors. Point-of-care testing can be used in many settings (for example, primary care, community clinics, and rural communities) and avoids traumatic blood draws in pediatric populations. Advantages include convenience, avoidance of specimen transport, minimal sample volumes, simple analytic processes, and immediate results. By improving access to kidney testing for underserved populations, POCT may have an important role in specific regions (**Table 1**).

Individual Risk Assessment for Kidney Failure in Persons With CKD

In persons with CKD G3 to G5, we recommend using an externally validated risk equation to estimate the absolute risk for kidney failure (1A, Recommendation 2.2.1, **Table 1**) (41–45). Among several existing validated risk prediction equations, the most validated is the Kidney Failure Risk Equation. Initially developed in 8400 Canadians, it has been externally validated in more than 2 million persons from 60 cohorts and 30 countries from nearly every continent demonstrating excellent discrimination (41, 42, 44, 45). Using patient's sex, age, uACR, and eGFR, the Kidney Failure Risk Equation provides 2- and 5-year estimates of the probability of kidney failure requiring KRT in those with eGFR less than 60 mL/min/1.73 m^2 (41–43, 45–47). The use of validated risk equations permits personalization and tailoring of treatment and care plans. Practice points describe a threshold 5-year kidney failure risk of 3% to 5% for the timing of nephrology referral, a 2-year risk of greater than 10% for the timing of multidisciplinary care, and a 2-year risk of 40% for method education and KRT preparation (dialysis access planning or referral for transplantation) (Practice Points 2.2.1 to 2.2.3, **Table 1**). We propose a shift from a GFR-based to a risk-based approach to CKD care and advanced care planning (Practice Points 2.3.1 to 2.3.2, **Table 1**) (**Appendix Figure 1**, available at Annals.org).

Delaying CKD Progression and Managing Its Associated Complications

Sodium–Glucose Cotransporter-2 Inhibitors

We recommend treating patients with type 2 diabetes, CKD, and an eGFR greater than 20 mL/min/1.73 m^2 with a sodium–glucose cotransporter-2 inhibitor (SGLT2i) (1A, Recommendation 3.7.1, **Table 1**) (**Appendix Figure 2**, available at Annals.org) (51). We now also offer more general recommendations for use of SGLT2i to include many adults with CKD who do not have diabetes, based on robust evidence (**Table 1**) (52). We recommend treating adults with CKD with an SGLT2i for eGFR greater than 20 mL/min/1.73 m^2 with uACR greater than 200 mg/g (>20 mg/mmol), or heart failure, irrespective of level of albuminuria (1A, Recommendation 3.7.2, **Table 1**).

Several large, placebo-controlled randomized controlled trials clearly show that SGLT2i, regardless of diabetes status, level of GFR, or cause of kidney disease, substantially reduce the risk for kidney failure, acute kidney injury, and hospitalization for heart failure, and also moderately reduce the risk for cardiovascular death and myocardial infarction in persons with and without CKD (52, 53). These benefits are summarized in a collaborative meta-analysis including 13 trials with just more than 90 000 randomized participants in comparison with placebo. Those allocated to an SGLT2i had a 37% reduction in the risk for kidney disease progression and a 23% reduction in the risk for acute kidney injury regardless of diabetes status (52).

The recommendation for use of SGLT2i in adults with eGFR 20 to 45 mL/min/1.73 m^2 with uACR less than 200 mg/g (<20 mg/mmol) was graded as a 2B (Recommendation 3.7.3, **Table 1**). Some uncertainty remains about the effects specifically on kidney disease progression in persons without diabetes who have CKD and uACR less than 200 mg/g (<20 mg/mmol), which led to a different grading of the recommendation for that population and outcome. EMPA-KIDNEY (Study of Heart and Kidney Protection with Empagliflozin) was the key trial to assess effects in persons with CKD at risk for progression with uACR less than 200 mg/g (<20 mg/mmol) and found evidence of interaction by uACR status for its primary outcome (trend $P = 0.02$) (54). Relative effects on the categorical outcome seemed to be larger in persons with higher levels of albuminuria. The slow rate of progression and small number of outcomes in the subgroup with normal to mildly increased albuminuria (<30 mg/g, A1) limited the power for EMPA-KIDNEY to assess effects on the primary outcome in this subgroup.

Note that the benefit of cardiovascular outcomes and hospitalization risk occurs regardless of level of albuminuria. This recommendation and grading specifically refers to the use of SGLT2i in this population to attenuate progression of CKD.

Practice Points 3.7.1 to 3.7.3 (**Table 1**) note that once initiated, it is reasonable to continue an SGLT2i even if the eGFR falls below 20 mL/min/1.73 m^2, unless not tolerated or KRT is initiated. Withholding SGLT2i during times of prolonged fasting, surgery, or critical illness (when persons may be at greater risk for ketosis) is reasonable. We highlight that SGLT2i initiation or use does not necessitate alteration of frequency of CKD monitoring, given that there is an expected reversible dip in eGFR, which is not an indication to discontinue therapy.

Hyperuricemia

We recommend that persons with CKD and symptomatic hyperuricemia should be offered uric acid–lowering intervention (1C, Recommendation 3.14.1, Table 1). There is strong evidence for uric acid lowering in persons with tophaceous gout, radiographic damage due to gout, or frequent gout flares, some of whom also had CKD, as identified by the American College of Rheumatology (55). We suggest not using agents to lower serum uric acid in persons with CKD and asymptomatic hyperuricemia to delay CKD progression (2D, Recommendation 3.14.2, Table 1). Despite observational studies implicating elevated serum uric acid levels in the progression of CKD, data from a 2017 Cochrane systematic review do not support treatment in the absence of symptoms (56). Since then, 3 further large, important randomized controlled trials focusing on the kidney benefits of lowering of asymptomatic hyperuricemia in persons with CKD have yielded negative results (57–59).

Statin Use

In accordance with the KDIGO guideline for lipid management (60) and because of increased risk for cardiovascular disease with CKD, we recommend treatment with a statin or statin–ezetimibe combination in adults aged 50 years or older not treated with chronic dialysis or kidney transplantation with eGFR less than 60 mL/min/1.73 m^2 (GFR categories G3a to G5) (1A, Recommendation 3.15.1.1, Table 1) and with eGFR 60 mL/min/1.73 m^2 or greater (GFR categories G1 to G2) (1B, Recommendation 3.15.1.2, Table 1) (1, 60). In persons with CKD, the same principles should be used to manage atherosclerotic risk as in persons without CKD, thus statins are recommended in patients aged 18 to 49 years with CKD not treated with chronic dialysis or kidney transplantation with known coronary artery disease, diabetes mellitus, prior ischemic stroke, or estimated 10-year incidence of coronary death or nonfatal myocardial infarction greater than 10% (2A, Recommendation 3.15.1.3, Table 1). Such effective treatments are often underused in persons with CKD presenting with acute coronary syndrome (61).

DISCUSSION

The KDIGO 2024 guideline navigates clinicians through the identification, improved evaluation, and targeted treatment and management of persons with CKD. Evaluation of CKD emphasizes the role of cystatin C for better accuracy of GFR, especially in clinical situations where creatinine falls short; the role of POCT in remote areas with limited access to a laboratory; and the shift to an individualized risk-based approach for prediction of kidney failure within a time frame of 2 to 5 years.

Sodium–glucose cotransporter-2 inhibitors are now recommended for persons with CKD with and without diabetes for kidney and cardiovascular benefit. Several large, placebo-controlled randomized controlled trials clearly show that SGLT2i, regardless of diabetes status, cause of kidney disease, or level of GFR, substantially reduce the risk for kidney failure, acute kidney injury, and hospitalization for heart failure, and moderately reduce the risk for cardiovascular death and myocardial infarction in persons with and without CKD. Likewise, statins have a role to prevent cardiovascular disease in patients with CKD.

The cost of new diagnostics and therapies may appear to be a barrier for their widespread implementation. The higher cost of cystatin C is believed to be mitigated by an evidence-driven approach to its use, which reduces misdiagnosis, improves accurate dosing of medications, and reduces adverse events due to medication errors, thus leading to a return on investment, and ultimately cost reduction through economies of scale. Costs of SGLT2i will likely be offset by delay or avoidance of CKD progression and benefits beyond kidney outcomes.

Limitations of this guideline include the gaps in the evidence base to inform the diagnostic testing strategies, optimal combination and timing of therapies, decision making, and processes of care. Thus, general recommendations include improved and broader clinical trial participation, especially of those who have been excluded most during the past decades (for example old, young, pregnant and lactating persons, and persons with advanced CKD). This will necessitate a change in attitude by both clinicians, patients, researchers, and funders. Representation of CKD in cardiovascular drug trials is still low, and exclusion of persons with CKD from these trials has even increased from 66% to 79% since 2000 (44). A further limitation is that studies do not account sufficiently for cause of CKD, sex, gender, age, socioeconomic status, and uACR in all cohorts and involve persons with CKD throughout the research process. The use of novel study designs and scientific methods, such as causal inference techniques, should be considered more and encouraged to move beyond the assessment of pure associations and to test new and multiple interventions in persons with CKD across the life cycle.

In conclusion, important evidence-based recommendations of this guideline include the use of more cystatin C, especially in the form of a GFR estimating equation based on both creatinine and cystatin C to increase accuracy of GFR values; use of risk-based approach using validated prediction equations (for example, Kidney Failure Risk Equation) for the risk for kidney failure to facilitate navigation of care and management; use of POCT in remote areas without a near laboratory; use of SGLT2i for persons with CKD regardless of diabetes or type of kidney disease to retard progression; use of uric acid–lowering medication in persons with CKD only if they are symptomatic; and use of statins for persons with CKD aged older than 50 years.

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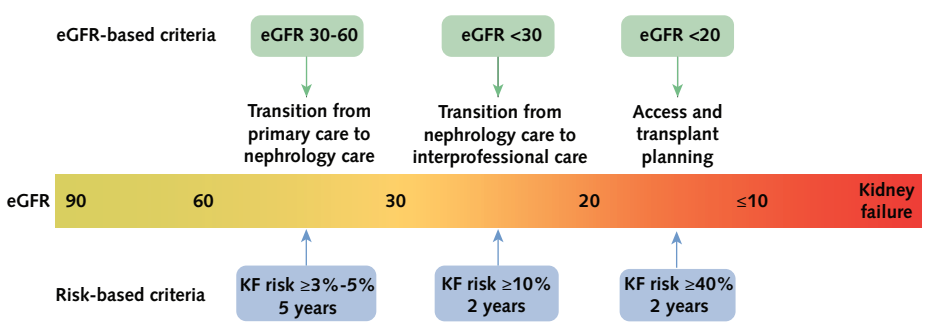
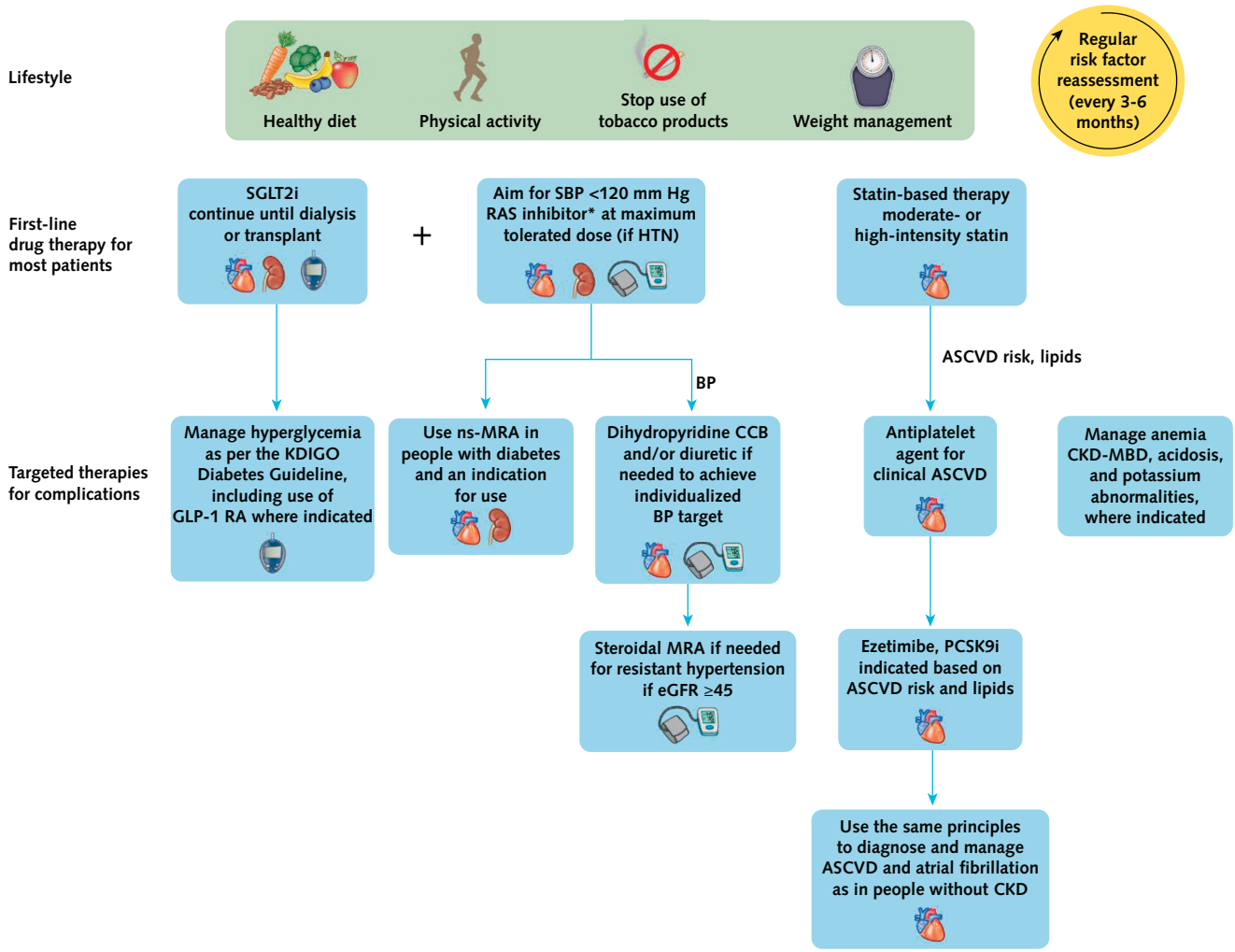
Appendix Figure 1. Transition from an eGFR-based to a risk-based approach to chronic kidney disease care.



eGFR = estimated glomerular filtration rate; KF = kidney failure. Reproduced from Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group. KDIGO 2024 clinical practice guideline for the evaluation and management of chronic kidney disease. Kidney Int. 2024;105:S117-S314; with permission.

Appendix Figure 2. Holistic approach to CKD treatment and risk modification.



ASCVD = atherosclerotic cardiovascular disease; BP = blood pressure; CCB = calcium-channel blocker; 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; with permission.

* Angiotensin-converting enzyme inhibitor or angiotensin II receptor blocker should be first-line therapy for BP control when albuminuria is present; otherwise dihydropyridine 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.