KDIGO 2024 CLINICAL PRACTICE GUIDELINE FOR THE EVALUATION AND MANAGEMENT OF CHRONIC KIDNEY DISEASE

Summary of recommendation statements and practice points

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).

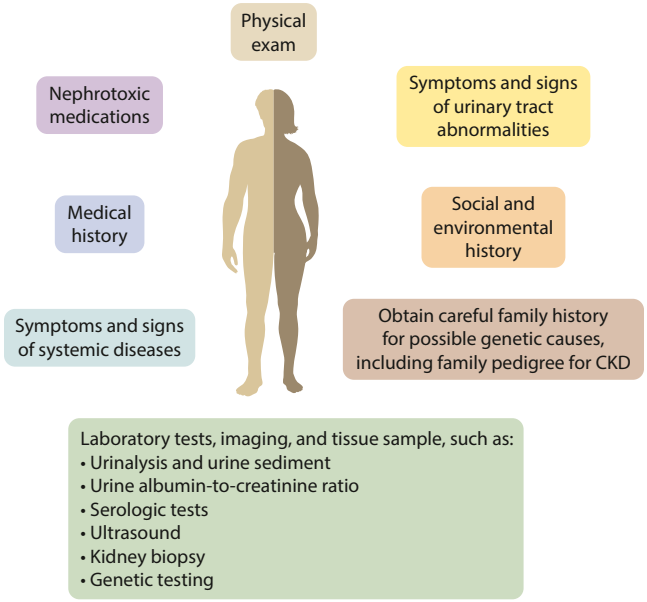

Figure 8| Evaluation of cause of chronic kidney disease (CKD).

Practice Point 1.1.4.2: Use tests to establish a cause based on resources available (Table 6[22,98-100]).

Table 6| Guidance for the selection of additional tests for evaluation of cause

Test category	Examples	Comment or key references
Imaging	Ultrasound, intravenous urography, CT kidneys ureters bladder, nuclear medicine studies, MRI	Assess kidney structure (i.e., kidney shape, size, symmetry, and evidence of obstruction) for cystic disease and reflux disease. Evolving role of additional technologies (e.g., 3D ultrasound)
Kidney biopsy	Ultrasound-guided percutaneous	Usually examined by light microscopy, immunofluorescence, and electron microscopy, and, in some situations, may include molecular diagnostics Used for exact diagnosis, planning treatment, assessing activity and chronicity of disease, and likelihood of treatment response; may also be used to assess genetic disease
Laboratory tests: serologic, urine tests	Chemistry including acid-base and electrolytes, serologic tests such as anti-PLA2R, ANCA, anti-GBM antibodies Serum-free light chains, serum, and urine protein electrophoresis/immunofixation Urinalysis and urine sediment examination	Refer to *KDIGO 2021 Clinical Practice Guideline for the Management of Glomerular Diseases*[22] Increasing recognition of the role of light chains in kidney disease even in the absence of multiple myeloma (monoclonal gammopathy of renal significance [MGRS])[98] Presence of persistent hematuria or albuminuria is critical in determining differential diagnosis
Genetic testing	*APOL1, COL4A3, COL4A4, COL4A5, NPHS1, UMOD, HNF1B, PKD1, PKD2*	Evolving as a tool for diagnosis, increased utilization is expected. Recognition that genetic causes are more common and may present without classic family history[99,100]

ANCA, antineutrophil cytoplasmic antibody; *APOL1*, apolipoprotein 1; *COL4A*, type IV collagen alpha chain; CT, computed tomography; GBM, glomerular basement membrane; *HNF1B*, hepatocyte nuclear factor 1B; MRI, magnetic resonance imaging; *NPHS1*, congenital nephrotic syndrome; *PKD1*, polycystic kidney disease-1; *PKD2*, polycystic kidney disease-2; PLA2R, M-type phospholipase A2 receptor; *UMOD*, uromodulin.

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).

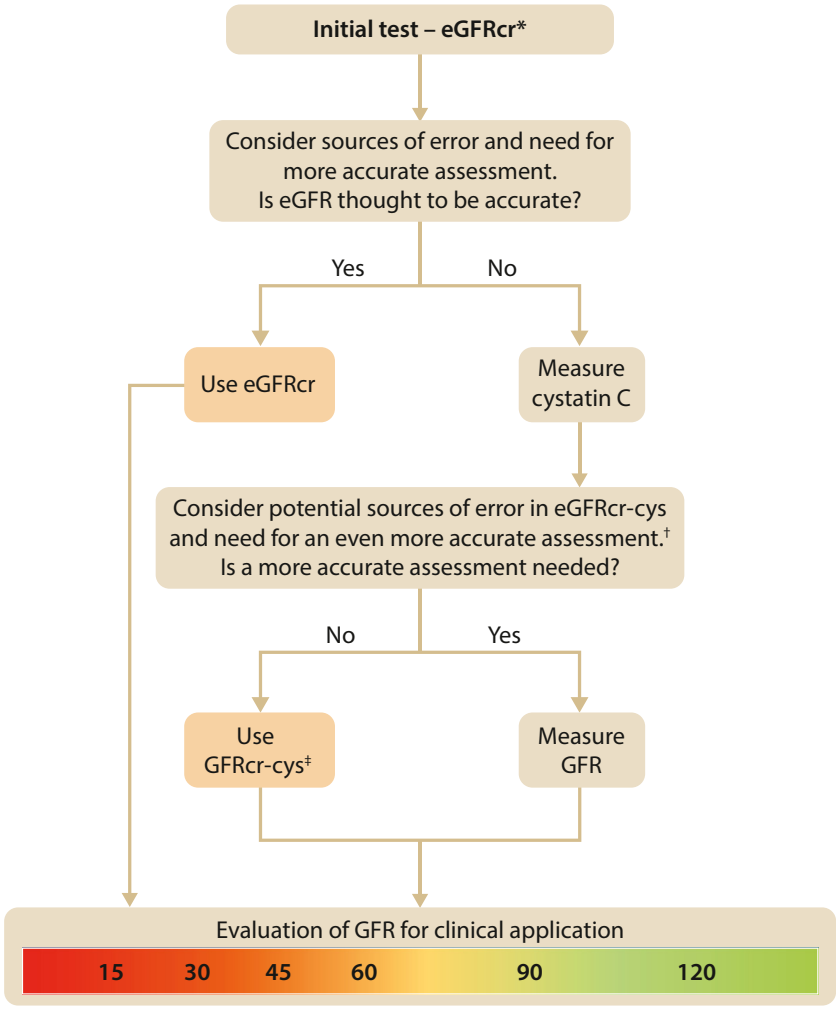

Figure 11 | Approach to glomerular filtration rate (GFR) evaluation using initial and supportive tests. The algorithm describes the approach to the evaluation of GFR. The approach uses initial and supportive testing to develop a final assessment of true GFR and to apply it in individual decision-making. The initial test for the evaluation of GFR is creatinine-based estimated GFR (eGFRcr), which will be available for most people because creatinine is measured routinely as part of the basic metabolic panel. If eGFRcr is expected to be inaccurate, or if a more accurate assessment of GFR is needed for clinical decision-making, such as diagnosis or staging of chronic kidney disease or drug dosing, then, if available, cystatin C should be measured, and creatinine and cystatin C–based estimated GFR (eGFRcr-cys) should be estimated. If eGFRcr-cys is expected to be inaccurate, or if an even more accurate assessment of GFR is needed for clinical decision-making, then, if available, GFR should be measured using plasma or urinary clearance of exogenous filtration markers. *Initial test may be estimated GFR by cystatin C (eGFRcys or eGFRcr-cys) in otherwise healthy populations with changes in creatinine generation due to non-GFR determinants such as changes in muscle mass or creatinine secretion or extrarenal elimination due to the use of specific medications. †Sources of error in eGFRcr-cys include very low muscle mass or very high levels of inflammation, high catabolic states, or exogenous steroid use. ‡Consider eGFRcys rather than eGFRcr-cys in otherwise healthy populations with decreased creatinine generation due to reduced muscle mass or decreased creatinine secretion or extrarenal elimination due to the use of specific medications.

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[127-142]) *(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).

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.

Table 8 | 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[127]	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[128]	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 comorbid illness.
	Class III obesity[a,b]	Non-GFR determinants of SCr and SCys	eGFRcr-cys demonstrated to be most accurate.
Lifestyle	Smoking[129-131]	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	
	Keto diets	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.
	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,132-137]	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,138,139]	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,79,140,141]	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[142]	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; GFR, glomerular filtration rate; mGFR, measured glomerular filtration rate; SCr, serum creatinine; SCys, serum cystatin C.

[a]Data summarized in Adingwupu *et al.*[149]

[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 measured glomerular filtration rate (mGFR) to evaluate this directly.

Table 9 | Comparison of estimated GFR and measured GFR

Estimated GFR by SCr and/or cystatin C	Measured GFR
Inexpensive and easy to implement	More expensive, more time-consuming, and invasive
Widely available and may also be used at point of care, easily repeatable	Only available in certain centers Methods to measure that do not require urine collections are available (i.e., plasma clearance) Most protocols require repeat blood samples potentially over a long duration Microsampling tests by fingerpick enable point-of-care testing. Testing has been described, but not routinely performed
Not sufficiently accurate and precise for all clinical situations	Accurate for GFR in all situations and across the GFR range. Requires individualized protocols
Lags behind changes in GFR	Able to identify early changes in GFR
Subject to non-GFR determinant confounding	Less influenced by non-GFR determinants

GFR, glomerular filtration rate; SCr, serum creatinine.

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 to ensure accuracy and reliability when assessing GFR using creatinine and cystatin C.

Table 11 | Implementation standards to ensure accuracy and reliability of GFR assessments using creatinine and cystatin C

- Report eGFR in addition to the serum concentrations of filtration markers using validated equations.
- Report eGFR rounded to the nearest whole number and relative to a body surface area (BSA) of 1.73 m^2 in adults using the units ml/min per 1.73 m^2.
- Reported eGFR levels <60 ml/min per 1.73 m^2 should be flagged as being low.
- When reporting levels of filtration markers, report:
  - (i) SCr concentration rounded to the nearest whole number when expressed as standard international units (μmol/l) and rounded to the nearest 100th of a whole number when expressed as conventional units (mg/dl);
  - (ii) serum cystatin C concentration rounded to the nearest 100th of a whole number when expressed as conventional units (mg/l).
- Measure filtration markers using a specific, precise (coefficient of variation [CV] <2.3% for creatinine and <2.0% for cystatin C) assay with calibration traceable to the international standard reference materials and desirable bias (<3.7% for creatinine and <3.2% for cystatin C) compared with reference methodology (or appropriate international standard reference method group target in external quality assessment [EQA] for cystatin C).
- Use an enzymatic method to assay creatinine, where possible.
- Separate serum/plasma from red blood cells by centrifugation within 12 hours of venipuncture.
- When cystatin C is measured, measure creatinine on the same sample to enable calculation of eGFRcr-cys.

eGFR, estimated glomerular filtration rate; eGFRcr-cys, estimated glomerular filtration rate based on creatinine and cystatin C; GFR, glomerular filtration rate; SCr, serum creatinine.

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.

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, 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).

Table 16 | Factors causing biological variation in urine albumin or urine protein

Factor	Falsely elevated ACR or PCR	False decrease in ACR or PCR
Variability in urine albumin or protein		
Hematuria	Increases albumin and protein in the urine	
Menstruation	Increases albumin and protein in the urine	
Exercise[259]	Increases albumin and protein in the urine	
Infection[260,261]	Symptomatic urinary infection can cause production of protein from the organism	
Nonalbumin proteins		Other proteins may be missed by albumin reagent strips
Variability in urinary creatinine concentration		
Biological sex	Females have lower urinary creatinine excretion, therefore higher ACR and PCR	Males have higher urinary creatinine excretion, therefore lower ACR and PCR
Weight[73,160]	Low urinary creatinine excretion consistent with low weight can cause high ACR or PCR relative to timed excretion	High urinary creatinine excretion consistent with high weight can cause low ACR or PCR relative to timed excretion
Changes in creatinine excretion	Lower urinary creatinine excretion with AKI or low-protein intake	High urinary creatinine excretion with high-protein intake or exercise

ACR, albumin-to-creatinine ratio; AKI, acute kidney injury; PCR, protein-to-creatinine ratio.

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 to ensure accuracy and reliability of the findings when assessing urine samples.

Table 17 | Implementation standards to ensure accuracy and reliability of urine samples

- Samples for albumin measurement analyzed fresh or stored at 4 °C for up to 7 days
- Samples for albumin measurement should not be stored frozen at −20 °C
- Report ACR in untimed urine samples in addition to urine albumin concentration rather than the concentrations alone
- Reporting to 1 decimal place for ACR whether mg/mmol or mg/g
- Analytical CV of methods to measure urine albumin should be <15%.

ACR, albumin-to-creatinine ratio; CV, coefficient of variation.

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.

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.

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).

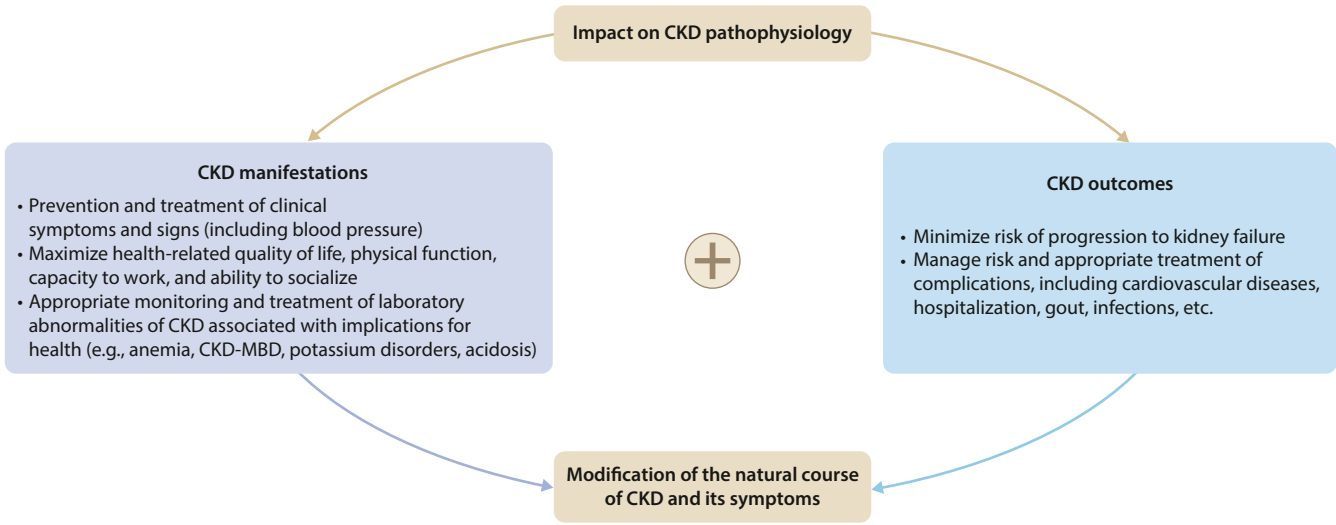

Figure 17 | Chronic kidney disease (CKD) treatment and risk modification. CKD-MBD, chronic kidney disease-mineral and bone disorders.

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 of tobacco products

[No specific recommendations or practice points]

3.2.2 Physical activity and optimum weight

The Work Group concurs with all the recommendation and practice points relating to physical activity from the KDIGO 2022 Clinical Practice Guideline for Diabetes Management in Chronic Kidney Disease[23] and considers that they should extend to all adults with CKD. We draw attention to the following statements:

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

The Work Group concurs with the following recommendation from KDIGO 2022 Clinical Practice Guideline for Diabetes Management in Chronic Kidney Disease[23] and the KDIGO 2021 Clinical Practice Guideline for the Management of Blood Pressure in Chronic Kidney Disease.[21]

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

The Work Group concurs with the KDIGO 2021 Clinical Practice Guideline for the Management of Blood Pressure in Chronic Kidney Disease, which encourages individualized BP targets and the use of agents according to age, coexistent CVD, and other comorbidities; risk of progression of CKD; and tolerance to treatments.[21] We highlight the following guidance:

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.

The Work Group concurs with the KDIGO 2021 Clinical Practice Guideline for the Management of Blood Pressure in Chronic Kidney Disease, and we highlight the following guidance[21]:

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.

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 for specific recommendations, practice points, and research recommendations.

3.6 Renin-angiotensin system inhibitors

The Work Group highlights recommendations from the KDIGO 2021 Clinical Practice Guideline for the Management of Blood Pressure in Chronic Kidney Disease and selected practice points for treatment with RASi from the KDIGO 2021 Clinical Practice Guideline for the Management of Blood Pressure in Chronic Kidney Disease[21] and the KDIGO 2022 Clinical Practice Guideline for Diabetes Management in Chronic Kidney Disease.[23] The Work Group considers several recommendations to apply even in the absence of high BP and has adapted the recommendations from the BP guideline to remove this requirement. Key recommendations and practice points are highlighted:

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)

The Work Group concurs with the KDIGO 2022 Clinical Practice Guideline for Diabetes Management in Chronic Kidney Disease, which stated: "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)*."[23] However, in the present guideline, we offer a more general 1A recommendation for adults with CKD. We also highlight practice points from the KDIGO Diabetes guideline for diabetes management in CKD, which are also relevant for people with CKD without diabetes:

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)

The Work Group highlights a key recommendation and practice points from the KDIGO 2022 Clinical Practice Guideline for Diabetes Management in Chronic Kidney Disease.[23]

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).

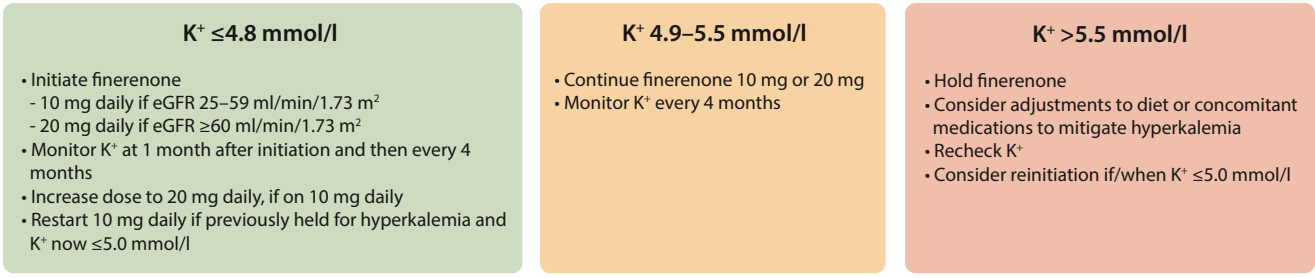

Figure 26 | Serum potassium monitoring during treatment with a nonsteroidal mineralocorticoid receptor antagonist (MRA) (finerenone). Adapted from the protocols of Finerenone in Reducing Kidney Failure and Disease Progression in Diabetic Kidney Disease (FIDELIO-DKD) and Finerenone in Reducing Cardiovascular Mortality and Morbidity in Diabetic Kidney Disease (FIGARO-DKD). The Work Group considers these potassium thresholds to be conservative, and it may be considered appropriate to continue MRAs in people with potassium of 5.5–6.0 mmol/l. This algorithm could be used for steroidal MRA. The US Food and Drug Administration (FDA) has approved initiation of $K^+ < 5.0$ mmol/l. This figure is guided by trial design and the FDA label and may be different in other countries. Serum creatinine/estimated glomerular filtration rate (eGFR) should be monitored concurrently with serum potassium. Reproduced 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.[23]

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)

The Work Group highlights a key recommendation and practice point from the KDIGO 2022 Clinical Practice Guideline for Diabetes Management in Chronic Kidney Disease.[23]

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.

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 recommendations and practice points]

3.11.4 Managing hyperkalemia

[No recommendations and 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

The KDIGO 2012 Clinical Practice Guideline for Anemia in Chronic Kidney Disease will be updated in 2024.[437]

3.13 CKD-Mineral Bone Disorder (CKD-MBD)

The Work Group highlights the KDIGO 2017 Clinical Practice Guideline Update for the Diagnosis, Evaluation, Prevention, and Treatment of Chronic Kidney Disease–Mineral and Bone Disorder (CKD-MBD).[20] Please refer to this publication 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).

Dietary approaches.

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

The benefits of lowering LDL cholesterol using statin-based therapies on the risk of ASCVD are well established in people with and without CKD. There are clear recommendations on when to initiate such therapies set out in the KDIGO Clinical Practice Guideline for Lipid Management in Chronic Kidney Disease.[19] The Work Group concurs with all the recommendations in this guideline. In particular, we draw attention to:

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.

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.

Dietary approaches.

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).

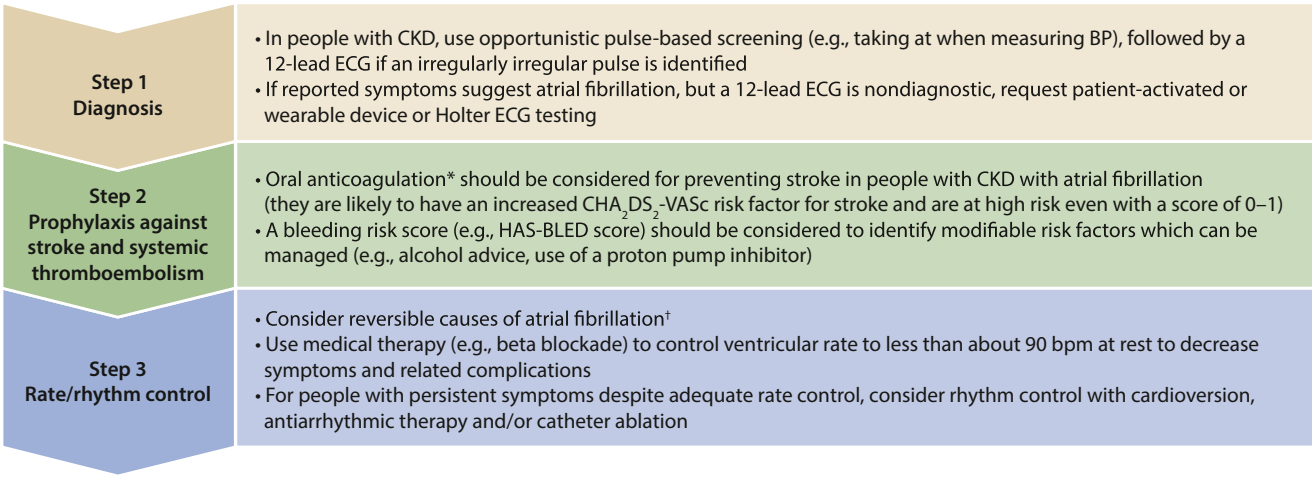

Figure 40| Strategies for the diagnosis and management of atrial fibrillation. *Consider dose adjustments necessary in people with chronic kidney disease (CKD). †The following has been recommended as a standard package for diagnostic evaluation of new atrial fibrillation: (i) a 12-lead electrocardiogram (ECG) to establish the diagnosis, assess ventricular rate, and check for the presence of conduction defects, ischemia, or structural heart disease; (ii) laboratory testing for thyroid and kidney function, serum electrolytes, and full blood count; and (iii) transthoracic echocardiography to assess left ventricular size and function, left atrial size, for valvular disease, and right heart size and function. BP, blood pressure; CHA_2DS_2-VASc, Congestive heart failure, Hypertension, Age ≥75 (doubled), Diabetes, Stroke (doubled), Vascular disease, Age 65 to 74, and Sex category (female); HAS-BLED, Hypertension, Abnormal liver/kidney function, Stroke history, Bleeding history or predisposition, Labile international normalized ratio (INR), Elderly, Drug/alcohol usage.

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).

	Dabigatran		Apixaban–Edoxaban–Rivaroxaban	
	No important bleeding risk and/or adequate local hemostasis possible: perform at trough level (i.e., ≥12 or 24 h after last intake)			
	Low risk	High risk	Low risk	High risk
CrCl ≥80 ml/min	≥24 h	≥48 h	≥24 h	≥48 h
CrCl 50–80 ml/min	**≥36 h**	**≥72 h**	≥24 h	≥48 h
CrCl 30–50 ml/min[a]	**≥48 h**	**≥96 h**	≥24 h	≥48 h
CrCl 15–30 ml/min[a]	No official indication	No official indication	**≥36 h**	**≥48 h**
CrCl <15 ml/min	No official indication for use There is no need for bridging with LMWH/UFH			

Figure 44 | Advice on when to discontinue non–vitamin K antagonist oral anticoagulants (NOACs) before procedures (low vs. high risk). The bold values deviate from the common stopping rule of ≥24-hour low risk, ≥48-hour high risk. Low risk is defined as a low frequency of bleeding and/or minor impact of a bleed. High risk is defined as a high frequency of bleeding and/or important clinical impact. Adapted from Heidbuchel H, Verhamme P, Alings M, et al. Updated European Heart Rhythm Association practical guide on the use of non–vitamin-K antagonist anticoagulants in patients with non-valvular atrial fibrillation: executive summary. *Eur Heart J.* 2017;38:2137–2149.[724] [a]Many of these people may be on lower dose of dabigatran (110 mg twice per day [b.i.d.]) or apixaban (2.5 mg b.i.d.), or have to be on the lower dose of rivaroxaban (15 mg QD) or edoxaban (30 mg QD). Dabigatran 110 mg b.i.d. has not been approved for use by the US Food and Drug Administration. CrCl, creatinine clearance, LMWH, low-molecular-weight heparin; UFH, unfractionated heparin. Reproduced from Turakhia MP, Blankestijn PJ, Carrero JJ, et al. Chronic kidney disease and arrhythmias: conclusions from a Kidney Disease: Improving Global Outcomes (KDIGO) Controversies Conference. *Eur Heart J.* 2018;39:2314–2325.[710]

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.

Medications and pregnancy.

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 College 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.

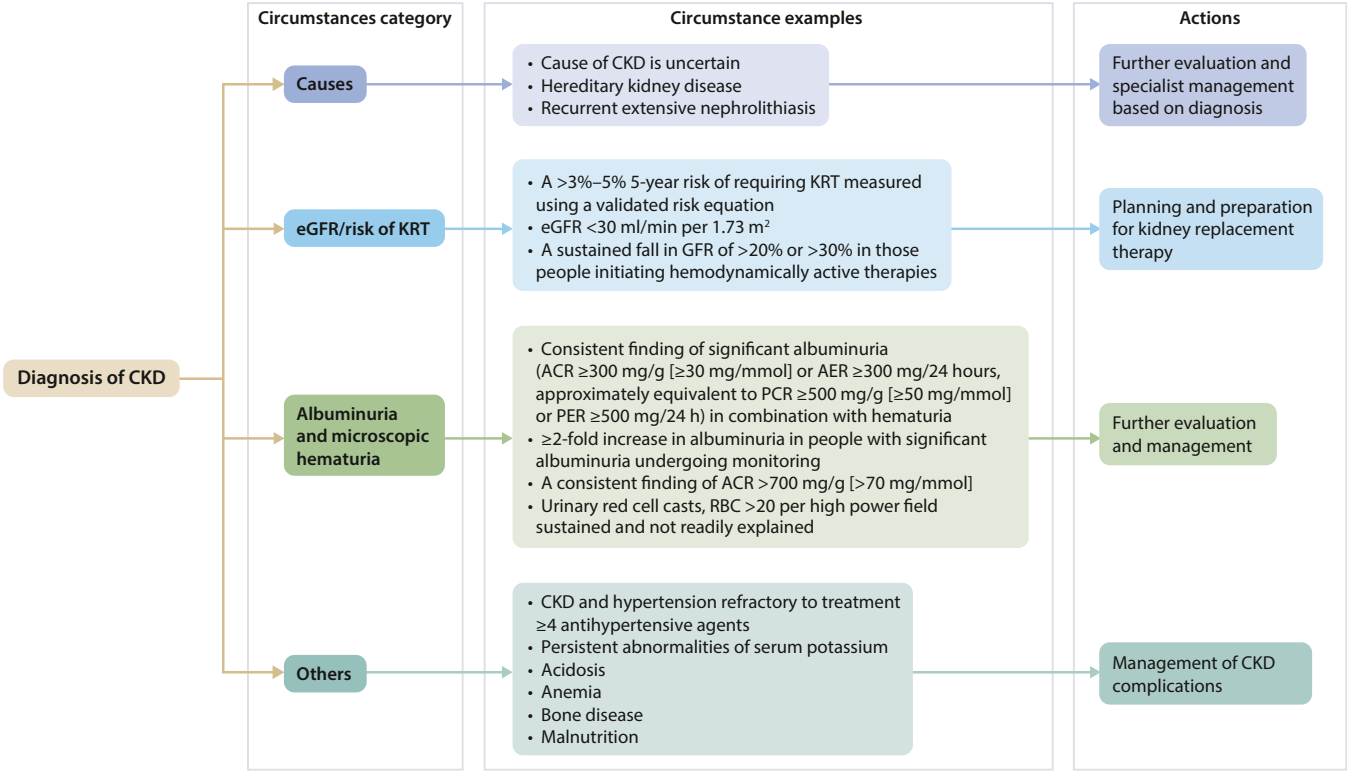

Figure 48 | Circumstances for referral to specialist kidney care services and goals of the referral. ACR, albumin-to-creatinine ratio; AER, albumin excretion rate; CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate; KRT, kidney replacement therapy; PCR, protein-creatinine ratio; PER, protein excretion rate; RBC, red blood cells.

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 recommendations and practice points]

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).

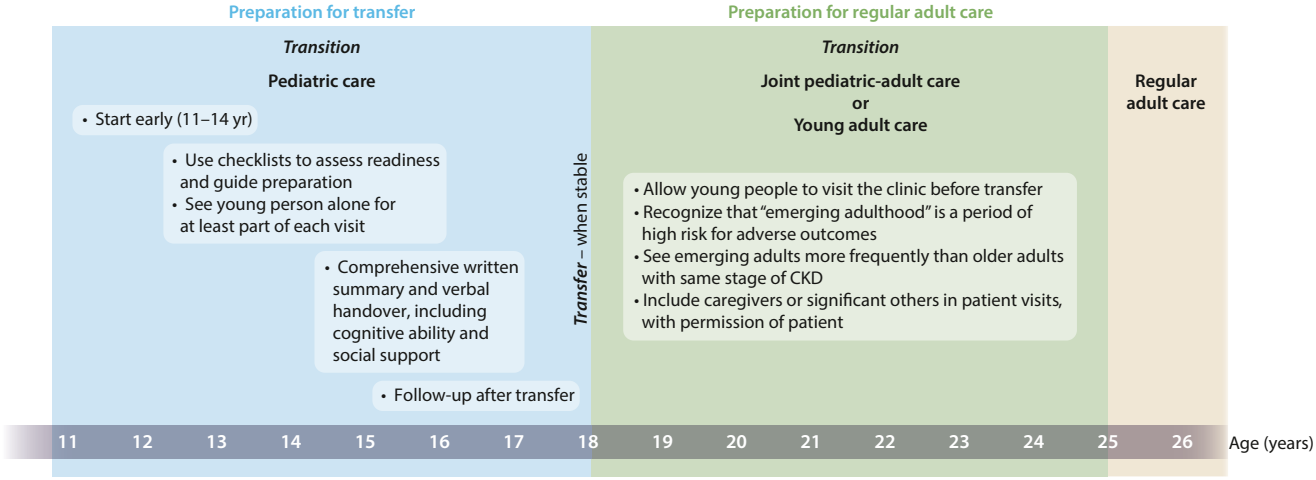

Figure 55 | The process of transition from pediatric to adult care in chronic kidney disease (CKD).

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).

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).

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).

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). This often but not invariably occurs in the GFR range between 5 and 10 ml/min per 1.73 m^2.

Table 41 | Indications for the initiation of dialysis

Symptoms or signs attributable to kidney failure (e.g., neurological signs and symptoms attributable to uremia, pericarditis, anorexia, medically resistant acid-based or electrolyte abnormalities, intractable pruritus, serositis, and acid-base or electrolyte abnormalities)
Inability to control volume status or blood pressure
Progressive deterioration in nutritional status refractory to dietary intervention, or cognitive impairment

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 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.