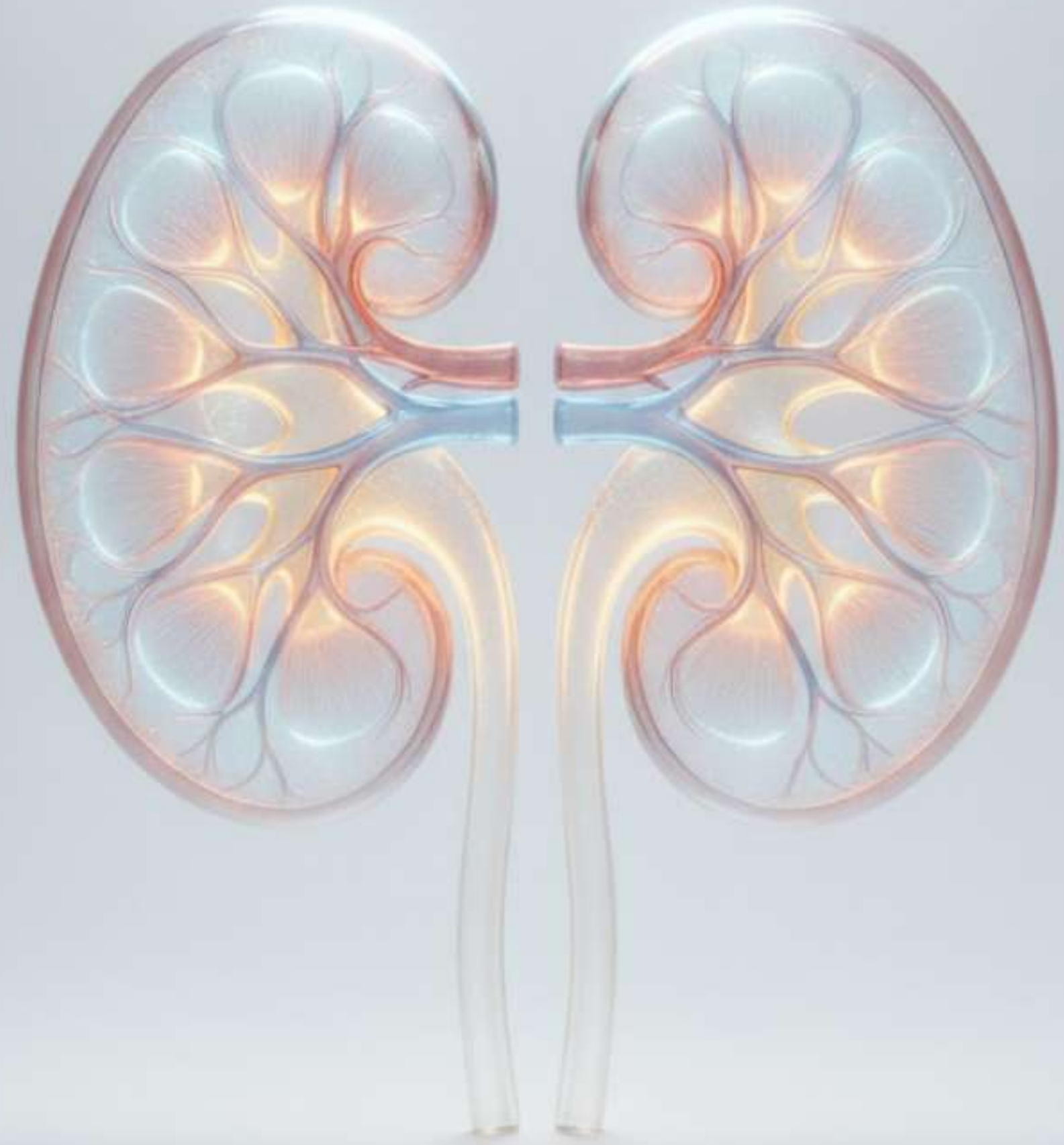
CHRONIC KIDNEY DISEASE

National Standard Treatment Guideline



Ministry of Health
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National Standard Treatment Guidelines

- Acid Peptic Disease
- Acute Anxiety
- Acute Pancreatitis
- Acute Psychosis
- Acute kidney Injury
- Arrhythmia
- Chronic Liver Disease
- Chronic Pancreatitis
- Chronic kidney disease
- Congenital Heart Diseases
- Dementia
- Depression
- Diabetes Mellitus Type 1
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- Hyponatremia
- Hypernatremia
- Hypokalemia
- Hyperkalemia
- Interstitial Lung Disease
- Liver Failure
- Obesity
- Obstructive Sleep Apnoea
- Osteoarthritis
- Ovarian Cancer
- Pneumonia
- Stroke
- Upper Gastrointestinal bleed
- Unstable Angina

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GUIDELINES DEVELOPMENT METHODOLOGY

The development of the Maldives Standard Treatment Guidelines (STGs) followed a structured, evidence-informed, and consensus-driven methodology adapted from internationally accepted guideline-development standards and the Delhi Society for Promotion of Rational Use of Drugs (DSPRUD) model. The process combined systematic evidence retrieval, critical appraisal, contextual adaptation, and multidisciplinary expert review to ensure feasibility, clinical relevance, and national ownership.

1. Determining Scope and Priority Conditions

Priority clinical conditions were identified through consultation with national programme managers, specialty clinicians, and health-system stakeholders. Selection criteria included: (i) major causes of morbidity and mortality, (ii) observed variation in clinical practice or prescribing patterns, (iii) potential to improve patient outcomes, and (iv) the feasibility of implementation across health-facility levels in Maldives. The final list of diseases reflected national epidemiology, service-delivery capacity, and essential-medicine availability.

2. Identification of Existing Evidence and Source Guidelines

A targeted search strategy was used to identify high-quality existing clinical guidelines. Searches were conducted across international guideline repositories (e.g., WHO, NICE, SIGN and other intergovernmental bodies, international and national guideline repositories, specialty societies and professional associations.

3. Quality Appraisal of Source Guidelines

Retrieved guidelines were screened for transparency of development, methodological rigour, clarity of recommendations, applicability to health-system reality, editorial independence. Guidelines were included if they met the Institute of Medicine (IOM) definition of a clinical guideline and addressed treatment or management of priority conditions. Guidelines that did not meet minimum quality standards, review articles, diagnostic criteria, or technical standards were excluded.

4. Adoption, Adaptation, and Contextualization

The guideline-development team employed an adopt–adapt–contextualize model:

- **Adoption**: High-quality recommendations that aligned with Maldivian health-system realities were retained without modification.
- **Adaptation**: Recommendations were modified when local considerations such as diagnostic capacity, medicine availability, workforce skills, referral pathways, or cost constraints affected feasibility.

- **Contextualization**: Where evidence was absent or inconclusive, conditional recommendations were formulated based on expert consensus, with explicit consideration of pragmatism, safety, and local workflows.
  Medicines were selected in alignment with the Maldives National Essential Medicines List (NEML), based on suitability, efficacy, safety, and availability.

5. Expert Consensus and Multidisciplinary Input

Draft recommendations were initially prepared by experts from the DSPRUD, India, providing a strong methodological foundation for the process. Building on this, a collaborative and participatory process brought together clinicians from internal medicine, paediatrics, obstetrics-gynaecology, surgery, emergency medicine, endocrinology, cardiology, general practitioners, and public health representing different levels of healthcare. Consensus was achieved through moderated discussions, iterative revisions, and resolution of divergent views. For topics lacking strong evidence, recommendations were derived from expert clinical judgment grounded in extensive practice experience.

6. Drafting, Peer Review, and Validation

Each guideline section was organized in a standard format including key clinical features, essential investigations, non-pharmacological management, pharmacological therapy (with step-up/step-down options where relevant), referral criteria, paediatric considerations, and follow-up requirements. Drafts were peer-reviewed by senior clinicians and national experts. Reviewer comments were systematically integrated to strengthen clarity, accuracy, and applicability.

7. Addressing Conflicts of Interest

All contributors declared the absence of conflicts of interest. Individuals with potential or perceived conflicts were excluded from authorship or decision-making roles.

8. Updating and Future Revisions

The STGs were conceptualized as a living document. Future updates will incorporate new scientific evidence, changes in essential-medicine availability, national programme priorities, and user feedback from clinicians. Periodic review cycles will ensure the continued relevance and reliability of recommendations.

9. Distinctive Features of the Guidelines

Developed through a collaborative process involving a large group of multidisciplinary experts from different levels of healthcare, the guidelines incorporate the following distinctive features:

- **Diagnostic Assumption and Confirmation**: While assuming that an initial diagnosis has been established by the healthcare provider, the guidelines provide essential information for confirming diagnoses. This includes a comprehensive overview of major signs and symptoms, descriptions of confirmatory tests, and clear guidance on practices that are prohibited, discouraged, or unreliable—promoting evidence-based medicine supported by relevant references.
- **Comprehensive Treatment Approach**: The guidelines offer a systematic, up-to-date framework for managing medical conditions across the continuum of care. They begin at the primary care level and extend to secondary and tertiary care, incorporating protocols for treatment response assessment and referral criteria as integral components.
- **Diverse Treatment Modalities**: Recommendations encompass both non-pharmacological and pharmacological interventions and surgical intervention where applicable, providing flexibility for individualized treatment plans. Cautionary notes are included where necessary to ensure safe and effective use of therapies.
- **Assessment and Referral Criteria**: Clear criteria and goals for evaluating patient response to treatment are provided, along with guidance on when referral to higher levels of care is warranted ensuring continuity and comprehensiveness in patient management.

ACKNOWLEDGEMENTS

The Government of the Republic of Maldives is committed to ensuring universal access to quality health services for all citizens. The Constitution of Maldives mandates the progressive realization of rights, including the right to good standards of health care for the population. In line with this national commitment, standardized quality health services are regarded as the foundation of a strong and equitable healthcare system.

This important work would not have been possible without the cooperation and support of many individuals and institutions. We express our sincere appreciation to the Honourable Minister of Health, Abdullah Nazim Ibrahim, for his leadership, commitment, and continuous guidance throughout the development process. We are grateful to WHO and ADB for their significant contribution, support, and technical assistance.

Our heartfelt gratitude is extended to the technical lead and editor, Dr. Sangeeta Sharma, Professor, Neuropsychopharmacology, IHBAS and President, Delhi Society for Promotion of Rational Use of Drugs (DSPRUD), and her team. We express our deepest appreciation to the Maldivian and DSPRUD experts and contributors who played a pivotal role in this process. Their technical expertise and dedication to adapt the standards to the Maldivian context have been instrumental in the development and finalization of these guidelines. The time, experience, generous sharing of knowledge and insights contributed by all parties have not only enriched the work but also have been invaluable in making these standards practical, locally acceptable, and aligned with the needs of the resident population.

It is important to acknowledge the immense efforts, involvement, timely coordination, collaboration, and dedication of the Quality Assurance and Regulation Division team who made it possible for these Clinical Treatment Guidelines to come into existence.

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CHRONIC KIDNEY DISEASE

QUICK REFERENCE GUIDE

Definitions & Types

- CKD: Abnormal kidney structure/ function ≥3 months with health implications.
- Types (by cause): Diabetic kidney disease, hypertensive nephrosclerosis, primary glomerular diseases, congenital/structural, tubulointerstitial, cystic/hereditary.
- CGA system: Cause + GFR (G1–G5) + Albuminuria (A1–A3) for staging and prognosis.

Causes, Risk Factors & Triggers

- Causes: Diabetes, hypertension, GN, reflux/obstruction, ischemic/ renovascular, cystic/hereditary, drug-induced.
- Risk factors: Age >60, CVD, family history, obesity, smoking.
- Triggers/accelerators: AKI, nephrotoxins(NSAIDs, aminoglycosides, contrast), dehydration, sepsis.

Evaluation for Diagnosis

- Clinical features: Often asymptomatic early; later—fatigue, dyspnea, edema, pruritus, anorexia, N/V, nocturia.
- Exam: BP ↑, pallor, edema, signs of CHF; pericarditis in advanced disease.
- Labs: SCr/eGFR, UACR, electrolytes, bicarbonate, calcium/phosphate, Hb.
- Urine: Dipstick + microscopy (protein, RBC casts).
- Imaging: Renal ultrasound (size, echotexture, obstruction).
- Confirm persistence ≥3 months; rule out AKI/reversible causes.

Classification / Severity

- GFR (mL/min/1.73 m^2): G1 ≥90*, G2 60–89*, G3a 45–59, G3b 30–44, G4 15–29, G5 <15/RRT. (*needs kidney damage evidence)
- Albuminuria (UACR mg/g): A1 <30, A2 30–300, A3 >300.
- Risk grid: Risk escalates with higher albuminuria at any G stage.

Differential Diagnosis (key look-alikes)

- AKI: abrupt change; often reversible.
- Renovascular disease: resistant HTN, asymmetric kidneys.
- Obstructive uropathy: hydronephrosis on US.
- Primary GN: proteinuria/hematuria, active sediment.
- Non-renal causes of low eGFR estimate: low muscle mass, lab error (repeat/confirm).

Management Goals

- Slow progression, control complications, reduce CV risk, maintain QoL, and prepare early for RRT when needed.

Management Principles

- Remove precipitants, treat emergencies, target the cause, optimize meds/doses, plan early for RRT, use multidisciplinary care.

Approach to Management (stepwise)

1. Classify CGA and assign risk.
2. Non-pharm for all (diet, BP targets, lifestyle, vaccines).
3. Pharm based on albuminuria, BP, diabetes, and complications.
4. Monitor and adjust (see schedule).
5. Refer by stage/risk; plan AVF and transplant early.

Non-Pharmacological

- Diet: sodium ≤60 mmol/day; moderate protein; limit phosphate/potassium if indicated; treat acidosis (bicarbonate).
- Lifestyle: stop smoking; ~150 min/wk activity; weight control.
- Hydration: individualized; routine restriction only in advanced CKD/ overload.
- Vaccines: influenza, pneumococcal, hepatitis B; COVID-19 boosters as per national guidance.
- Low-resource adaptations: rely on creatinine trends and BP if UACR delayed; prioritize ACEi/ARB + lifestyle; calcium binders if non-calcium unavailable (avoid hypercalcemia); use US over advanced imaging; peritoneal dialysis may be more feasible; structured referral thresholds.

PHARMACOLOGICAL THERAPY

Indication	Drug (examples)	Dose (adult)	Route / Duration	Key cautions
Albuminuria A2–A3 ± HTN	**ACEi/ARB** (ramipril, telmisartan, losartan, enalapril)	Ramipril 2.5–10 mg/day; Telmisartan 40–80 mg/day; Losartan 25–100 mg/day; Enalapril 2.5–20 mg/day	Oral, long-term	Monitor K^+/SCr; hold during AKI; avoid dual ACEi+ARB
Hypertension add-on	**CCB** (amlodipine)	5–10 mg/day	Oral, long-term	Edema
Volume overload	**Loop diuretics** (furosemide, torsemide)	Furosemide 20–160 mg/day; Torsemide 5–20 mg OD (max 100 mg/day)	Oral/IV, ongoing	Electrolytes, dehydration
T2D + CKD kidney/CV protection	**SGLT2i**	Dapagliflozin 10 mg OD; Empagliflozin 10 mg OD; Canagliflozin 100 mg OD	Oral, long-term	Start if eGFR ≥20; transient eGFR dip; sick-day rules
Glycemic base	**Metformin**	Per label if eGFR ≥30	Oral, long-term	Stop if eGFR <30/AKI
Anemia (after iron repletion)	**ESA** (epoetin, darbepoetin)	Epoetin 50–100 IU/kg SC weekly; Darbepoetin 0.45 µg/kg q1–2 wk	SC/IV, long-term	HTN, thrombosis; Hb ~10–11.5
Iron deficiency	**Oral/IV iron**	Ferrous sulfate 200 mg TDS; Iron sucrose 100–200 mg IV weekly; Ferric carboxymaltose 500–1000 mg (≤15 mg/kg), repeat ≥1 wk; max 1500 mg/course	Oral/IV, courses	Ferritin/TSAT; hypersensitivity (IV)
Hyperphosphatemia	**Binders**	Calcium carbonate 500–1000 mg TDS with meals; Sevelamer 800–1600 mg TDS	Oral, ongoing	Ca/PO_4 balance; prefer non-calcium if Ca high
Secondary HPT	**Calcitriol**	0.25–0.5 µg/day (titrate)	Oral, ongoing	HyperCa/PO_4
Metabolic acidosis	**Sodium bicarbonate**	500 mg–1 g TDS	Oral, until HCO_3^- ≥22	Sodium load/volume

Assessment of Response & Treatment Review

- At each visit: BP, volume status, adherence, side-effects, labs(SCr/eGFR, K^+, Hb; UACR quarterly).
- Before step-up/step-down: confirm adherence, salt intake, home BP log, recent AKI, drug interactions, and updated labs.
- If uncontrolled: intensify lifestyle, uptitrate ACEi/ARB (if tolerated), add CCB/diuretic; add SGLT2i; address complications.

Referral (tiered)

- Primary → Secondary: any persistent eGFR <60, A3 albuminuria, resistant HTN, recurrent AKI, suspected GN/ obstruction.
- Secondary → Tertiary/Nephrology: CKD Stage ≥3, rapid decline (>5 mL/min/ year), refractory complications, suspected systemic GN.
- RRT planning: AV fistula ≥6 months before expected dialysis; transplant referral when eGFR <30.

Complications (recognize & act)

- Early: HTN, anemia, mild acidosis, hyperkalemia, fatigue.
- Late: uremic symptoms, pulmonary edema, MBD, pericarditis, malnutrition, ESRD, major CV events.

Patient Education & Instructions

- Understand stage/trajectory.
- Self-management: BP/glucose, diet, fluids, meds, avoid nephrotoxins.
- Recognize warning signs: edema, ↓urine, dyspnea, palpitations, confusion.
- Reduce CV risk: quit smoking, activity, weight control.
- Prepare for RRT: timing, AVF planning, modality choices, transplant basics; lifelong immunosuppression if transplanted.
- Vaccinations (influenza, pneumococcal, hepatitis B; COVID-19 boosters per national guidance).
- Home monitoring & when to seek care.
- Shared decisions & advance care planning.

INTRODUCTION

Chronic Kidney Disease (CKD) affects about 10% of the global population with over 670 million people, making it a major public health issue. It is most often caused by diabetes, hypertension, and cardiovascular disease, with prevalence increasing in older adults. CKD is now among the top ten global causes of premature death, responsible for more than 1.5 million deaths annually, and contributes heavily to disability, healthcare costs, and cardiovascular risk.

In South and Southeast Asia, prevalence is among the world's highest, driven by metabolic diseases, aging populations, and lifestyle changes. CKD progresses silently, with early stages often asymptomatic but amenable to intervention. Blood pressure and glycemic control, proteinuria reduction, and avoiding nephrotoxins can slow progression and reduce complications. Without standardized care, patients present late with advanced disease, leading to worse outcomes and heavier demand for dialysis and transplant services. Standardized guidelines enable early detection, consistent risk stratification, and evidence-based management, improving survival and resource use.

Scope and Applicability

- Covers evaluation, risk stratification, prevention, and management of CKD in adults.
- Intended for primary care/general physicians, nephrologists, nurses, renal dietitians, diabetes educators, pharmacists, community health workers, and program/planning stakeholders.
- **Primary / Community Level**
  - Early detection: eGFR & albuminuria screening in at-risk groups.
  - Control of hypertension/diabetes; lifestyle counseling; avoid nephrotoxins.
  - Educate patients, identify warning signs, and refer when eGFR declines or complications appear.
  - In low-resource settings: rely on creatinine trends + clinical judgment.
- **Secondary / District Hospitals**
  - Full diagnostic evaluation: etiology, staging, anemia, mineral bone disorder.
  - Start renoprotective therapy (RAS blockade, correct acidosis).
  - Manage complications, plan for renal replacement access.
- **Tertiary / Specialist Care**
  - Advanced diagnostic workup and management of complex cases.
  - Dialysis initiation, vascular access, and transplant evaluation.
  - Coordinate long-term follow-up.

Adaptation for Limited Resources

- Address gaps: delayed albuminuria assays, limited imaging, few specialists, restricted drug/dialysis access.
- Use simplified algorithms and clear referral thresholds.
- Emphasize strong referral systems and cross-level communication to ensure timely care.

DEFINITION

CKD is defined as abnormality of kidney structure or function present for more than 3 months. Presence of GFR of less than 60 mL/min/1.73 m2, an albumin excretion rate of 30 mg/24 hours or greater and urine albumin–creatinine ratio (ACR) of 30 mg/g or greater for more than 3 months are diagnostic of CKD.

Kidney failure is the most advanced stage of chronic kidney disease and is defined as:

- GFR <15 mL/min/1.73 m^2, or
- Requirement for renal replacement therapy (RRT) - dialysis or transplantation.

At this stage, the kidneys are unable to maintain fluid, electrolyte, and metabolic balance sufficient to sustain life. Without initiation of RRT, kidney failure is incompatible with long-term survival (KDIGO 2024).

RISK FACTORS & TRIGGERS

Category	Examples	Impact
Major Risk Factors	• Diabetes mellitus • Hypertension	Leading global causes of CKD; drive both onset and progression
Other Medical Risks	• Cardiovascular disease • Older age (>60 years) • Family history of kidney disease	Increase vulnerability to kidney injury and accelerate decline
Lifestyle Risks	• Smoking - Obesity	Associated with faster progression, higher CV and renal risk
Triggers / Accelerators	• Nephrotoxic drugs (NSAIDs, • aminoglycosides, some antivirals, chemotherapy agents) • Radiographic contrast agents • Acute illness: dehydration, sepsis, volume depletion	Can cause acute kidney injury on CKD or accelerate progression

EVALUATION FOR DIAGNOSIS

Domain	Key Points
Symptoms	• Early CKD: asymptomatic • Later stages (uraemia): fatigue, dyspnoea, anorexia, nausea, vomiting, ankle oedema, pruritus, purpura, neuromuscular disturbances, nocturia
Signs on Examination	• Pallor (anaemia) • Nail dystrophy, purpura • Hypertension, cardiomegaly • Signs of CHF: pulmonary oedema, pleural effusion • Pericarditis (± pericardial effusion)
Laboratory Findings	• Elevated serum creatinine & urea (often detected during anaemia workup) • Biochemical abnormalities: hypocalcaemia, hyperphosphataemia, hyperkalaemia, metabolic acidosis (partially compensated) • Peripheral smear: normocytic, normochromic anaemia • Urinalysis: proteinuria, low fixed specific gravity
Imaging & Further Investigations	• Ultrasound: small, shrunken kidneys → ESRD • Normal/enlarged kidneys: diabetic nephropathy, polycystic kidney disease → consider renal biopsy if etiology unclear - Skeletal survey: features of renal osteodystrophy

CLASSIFICATION

CKD is classified by the degree of kidney function loss and albuminuria severity, using the KDIGO framework based on:

1. Cause
2. GFR category (G1–G5)
3. Albuminuria category (A1–A3)

KDIGO classification by GFR category and albuminuria Categories with Common Causes

GFR Category (G)	eGFR (mL/min/1.73 m²)	Description	Common Causes (with kidney damage or progression)
G1	≥90 (with evidence of kidney damage)	Normal or high	Diabetes, hypertension, glomerulonephritis, polycystic kidney disease, congenital anomalies
G2	60–89 (with evidence of kidney damage)	Mildly decreased	Same as G1; often early diabetic nephropathy, hypertensive nephrosclerosis

G3a	45–59	Mild–moderately decreased	Diabetes, hypertension, glomerulonephritis, chronic interstitial nephritis, obstructive uropathy
G3b	30–44	Moderately–severely decreased	Diabetes, hypertension, ischemic nephropathy, chronic GN, reflux nephropathy
G4	15–29	Severely decreased	Advanced diabetic nephropathy, hypertensive kidney disease, chronic GN, hereditary kidney disease
G5	<15 (or dialysis-dependent)	Kidney failure	End-stage renal disease due to diabetes, hypertension, GN, polycystic kidney disease, obstructive/reflux nephropathy

Albuminuria Category (A)	UACR (mg/g) [mg/mmol]	Description	Common Causes / Examples
A1	<30 (<3)	Normal to mildly increased	Normal physiology; transient proteinuria (fever, exercise, dehydration); early diabetes or hypertension (before overt kidney damage)
A2	30–300 (3–30)	Moderately increased (microalbuminuria)	Early diabetic nephropathy, hypertensive nephrosclerosis, obesity/metabolic syndrome, early glomerulonephritis, chronic interstitial nephritis
A3	>300 (>30)	Severely increased (macroalbuminuria)	Advanced diabetic kidney disease, glomerulonephritis (IgA, post-infectious, lupus), nephrotic syndrome, amyloidosis, advanced hypertensive nephrosclerosis

KDIGO CGA risk stratification "heat map" 2024: Prognosis of CKD by GFR and Albuminuria Category

Combined staging based on GFR and albuminuria improves risk stratification of CKD patients. Higher stages have worse prognosis.

Table . KDIGO prognosis for chronic kidney disease (CKD) based on glomerular filtration rate (GFR) and albuminuria.

GFR Category (mL/min/1.73 m²)	A1: <30 mg/g (Normal to mildly increased)	A2: 30–300 mg/g (Moderately increased)	A3: >300 mg/g (Severely increased)
G1: ≥90	Low risk	Moderate risk	High risk
G2: 60–89	Low risk	Moderate risk	High risk
G3a: 45–59	Moderate risk	High risk	Very high risk
G3b: 30–44	High risk	Very high risk	Very high risk
G4: 15–29	Very high risk	Very high risk	Very high risk
G5: <15	Very high risk	Very high risk	Very high risk

Note: The risk categories reflect both kidney disease progression and associated cardiovascular risk. Regular monitoring and individualized management are essential at each stage.

Interpretation of Prognosis:

- Low Risk: Stable disease; yearly monitoring.
- Moderately Increased Risk: Progression possible; close monitoring every 6–12 months.
- High Risk: Likely disease progression and cardiovascular events; monitor every 3–6 months.
- Very High Risk: Rapid progression expected; frequent monitoring (1–3 months), preparation for renal replacement therapy necessary.

DIFFERENTIAL DIAGNOSIS

Key alternatives or overlapping conditions to consider include the following. Distinguishing these relies on history, timing, urinalysis, imaging, and selective serologic or functional testing.

Condition	Key Features	How to Distinguish from CKD
Acute Kidney Injury (AKI)	Rapid rise in serum creatinine, oliguria/ anuria over days; often reversible	Recent onset, identifiable trigger (e.g., dehydration, sepsis, nephrotoxins), absence of chronic structural changes on imaging; improvement after correction of cause
Renovascular Disease (Renal artery stenosis)	Resistant hypertension, progressive decline in GFR, asymmetric kidneys, abdominal bruit	Imaging: Doppler US, CT/MR angiography; high renin states; unilateral or bilateral kidney asymmetry
Obstructive Uropathy	Post-renal block from stones, strictures, prostate enlargement; lower urinary symptoms	Ultrasound/CT: hydronephrosis, dilated collecting system; relief after decompression improves function
Primary Glomerular Diseases (e.g., IgA nephropathy, FSGS, lupus nephritis)	Proteinuria, hematuria, RBC casts, active urinary sediment	Serologic workup (ANA, ANCA, complements); renal biopsy confirms diagnosis
Diabetic Nephropathy	Long-standing diabetes, persistent albuminuria, declining GFR; often with retinopathy	History of diabetes, microvascular complications, typical progression pattern; biopsy if atypical features

COMPLICATIONS

Chronic kidney disease leads to multiple systemic complications (early and late). If these remain unchecked, it may accelerate loss of kidney function and culminate in progression to end-stage renal disease, necessitating renal replacement therapy.

Systemic Complications of Chronic Kidney Disease

Stage	Complications	Impact
Early Complications	• Hypertension • Anemia of chronic disease • Electrolyte disturbances (esp. hyperkalemia) • Mild metabolic acidosis - Proteinuria • Fatigue, reduced exercise tolerance	Often asymptomatic or subtle; if untreated, accelerate CKD progression and CV risk
Late Complications	• Uremic symptoms: nausea, pruritus, encephalopathy • Volume overload → pulmonary edema • Mineral & bone disorders (renal osteodystrophy) • Pericarditis • Malnutrition / wasting • End-stage renal disease (ESRD) • Cardiovascular disease: LVH, heart failure, MI, arrhythmia	Mark advanced kidney failure; major drivers of morbidity, mortality, and need for dialysis/transplant

RISK FACTORS FOR PROGRESSION OF CKD

Category	Risk Factors	Notes / Examples
Modifiable	• Poorly controlled diabetes mellitus • Hypertension • Persistent proteinuria • Smoking • Obesity • Recurrent episodes of AKI • Nephrotoxic drug use (NSAIDs, contrast, some antibiotics) • Hyperlipidemia • Metabolic acidosis • Inadequate follow-up / poor adherence	Targeted interventions (glycemic & BP control, RAS blockade, smoking cessation, weight loss, statins, bicarbonate therapy, medication adherence) can slow progression
Non-Modifiable	• Age >60 years • Male sex • Family history of CKD • Ethnicity: South Asians, African ancestry higher risk • Congenital/structural kidney disease (CAKUT, reflux nephropathy) • Genetic mutations (APOL1, PKD1/2, others)	Identify high-risk individuals for early screening, monitoring, and counseling

MANAGEMENT GOALS

- Slow progression of kidney disease.
- Control symptoms and complications (anemia, mineral bone disorder, acidosis, electrolyte imbalance, fluid overload).
- Reduce cardiovascular risk through BP, lipid, and glycemic control.
- Optimize quality of life with lifestyle measures (smoking cessation, diet, weight management).
- Plan early for renal replacement therapy (RRT) with timely education, referral, and vascular access preparation.

KEY PRINCIPLES OF MANAGEMENT

1. Address reversible factors: stop nephrotoxins, correct hypovolemia, treat infection/ obstruction, control hypertension, diabetes, and heart failure.
2. Treat emergencies promptly: severe hyperkalemia, pulmonary edema, metabolic acidosis, uremic encephalopathy, accelerated hypertension.
3. Target underlying cause: e.g., intensive glycemic control in diabetic nephropathy, RAAS blockade for proteinuria.
4. Optimize drug therapy: adjust renally cleared medications to avoid toxicity.
5. Plan early for RRT: dialysis or transplant evaluation, vascular access creation, patient/ family education.
6. Use multidisciplinary care:
   - Nephrologists: staging, renal-specific therapy, RRT planning.
   - Primary care: comorbidity management (diabetes, HTN, CVD).
   - Dietitians: nutrition (protein, sodium, phosphorus).
   - Diabetes educators: support for glycemic control.
   - Pharmacists: drug dose adjustment, interaction prevention.
   - Social support teams: adherence, access, psychosocial support

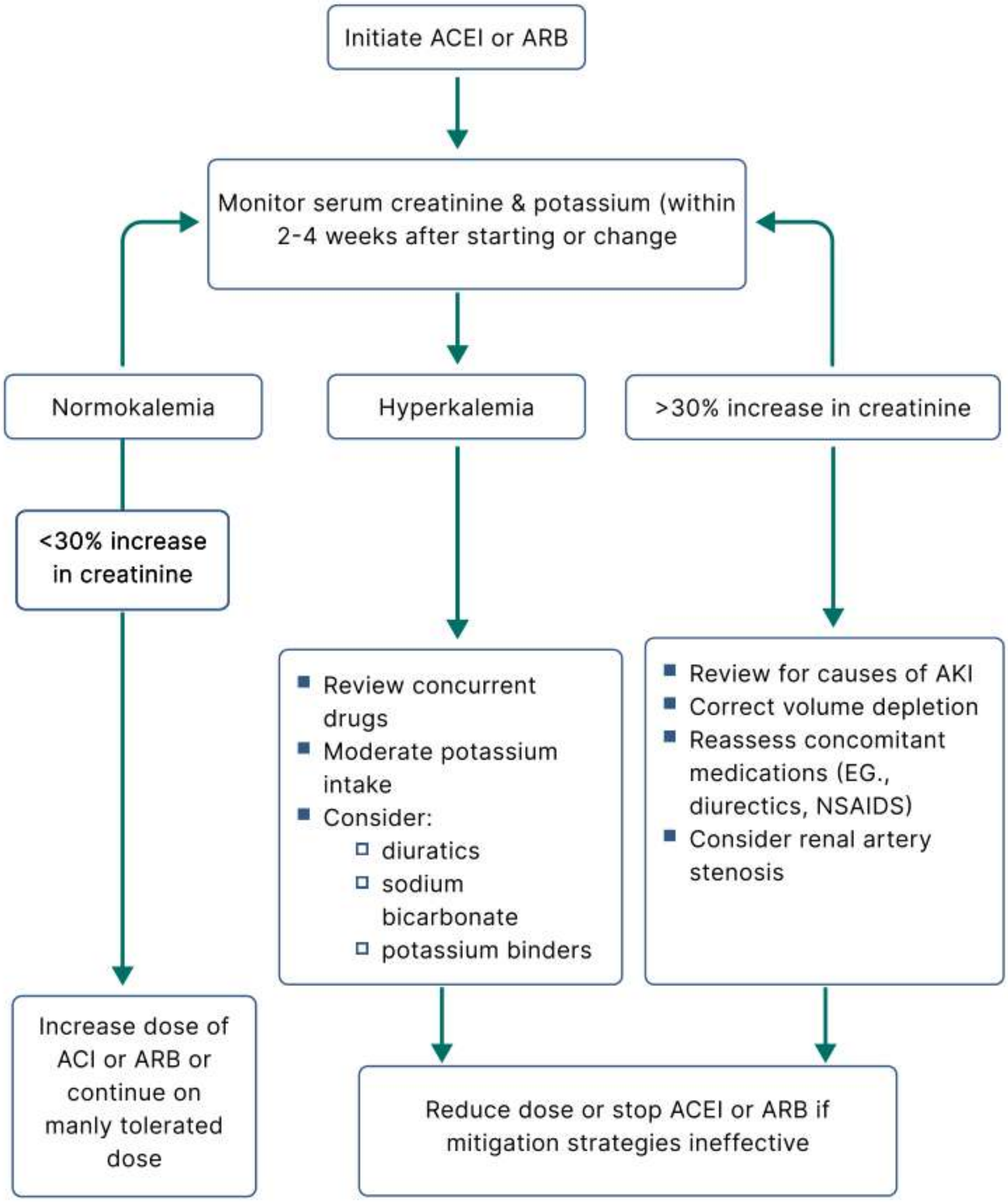

Fig 1. Approach for monitoring serum creatinine and potassium in patients initiated on angiotensin-converting enzyme inhibitors (ACEi) or angiotensin II receptor blockers (ARB). Reversible Causes of Renal Function Decline.

SLOWING THE RATE OF PROGRESSION OF CKD

In addition to treating the underlying cause of CKD, several interventions help slow disease progression:

A. Treat emergencies promptly: severe hyperkalemia, pulmonary edema, metabolic acidosis, uremic encephalopathy, accelerated hypertension.

B. Blood glucose control: Essential in patients with diabetes to prevent further renal injury.

C. Reduction of proteinuria: Minimize protein loss in urine to slow renal decline.

D. Use of ACE inhibitors (ACEIs) or ARBs (with caution if eGFR <30 or K+ >5.5) (Figure 1):

- For patients with diabetes and albuminuria but normal blood pressure, an ACE inhibitor or ARB in proteinuric CKD (e.g., Enalapril 5–20 mg/day orally). After initiation or dose increase, monitor blood pressure, serum creatinine, and potassium within 2–4 weeks. An initial creatinine rise of up to 25% within the first week is acceptable and not a reason to stop therapy. Continue therapy unless creatinine rises by more than 30% in that interval.

  Caution: Women of childbearing potential should use contraception while on ACEi/ARB and these agents should be stopped in those planning pregnancy or who become pregnant. Use only one renin-angiotensin system blocker at a time. Combining an ACEi with an ARB, or either with a direct renin inhibitor, is potentially harmful and should be avoided.

- If hyperkalaemia does not develop, ACEIs/ARBs can be continued even in advanced CKD stages as they provide long-term renal protection.

- Hyperkalemia from ACEi/ARB is often managed by measures to lower serum potassium (review concurrent drugs, dietary intake, consider diuretics, bicarbonate, or binders) rather than immediate dose reduction. Reduce or stop the agent only if there is symptomatic hypotension, persistent/refractory hyperkalemia despite mitigation, or when easing uremic symptoms is needed in advanced kidney failure (eGFR <15 mL/min/1.73 m^2).

- SGLT2i recommended for eGFR ≥20 in T2D with CKD start at lowest trial dose (dapagliflozin 10 mg, empagliflozin 10 mg, canagliflozin 100 mg). Caution: eGFR dip of a few mL/min is expected after starting; do not stop for small, reversible declines. If >30% fall, evaluate volume status, RAASi/diuretic dosing, intercurrent illness. Sick-day rules: hold during prolonged fasting, major surgery, or critical illness (DKA risk). Resume after recovery.(KDIGO 2024)

E. Dietary protein restriction (with dietitian input to avoid malnutrition) For details see non-pharmacological section below)

F. Management of chronic metabolic acidosis: Correction with alkali therapy Sodium bicarbonate 600 mg PO 4 times a day if plasma bicarbonate <20 mmol/L may help preserve renal function.

G. It is important to identify and address reversible factors contributing to renal dysfunction:

- Reduced renal perfusion: Due to hypovolaemia or hypotension.
- Exposure to nephrotoxic agents: Such as certain antibiotics, NSAIDs, or contrast media.
- Urinary tract obstruction: e.g., from stones, tumors, or prostatic hypertrophy.

NON-PHARMACOLOGICAL

Non-pharmacological management focuses on lifestyle and risk reduction

Domain	Measures	Targets
Dietary Adjustments	• Moderate Sodium restriction	≤60 mmol/day (avoid added salt, processed foods); helps BP and edema control
	• Protein restriction	• Moderate intake (avoid very high protein diets); balance with nutrition needs; • Early stages: 0.8 g/kg/day (high biological value protein preferred). • Advanced CKD: 0.6 g/kg/day. • Note: Once dialysis is initiated, protein intake should be increased to prevent malnutrition.
	• Phosphate restriction	~1000 mg/day; avoid milk, eggs, cola, processed foods; reduces vascular/soft tissue calcification
	• Potassium restriction	If moderate–severe CKD; avoid bananas, citrus, papaya, coconut water, melons
	• Correct metabolic acidosis	Sodium bicarbonate 600 mg PO 4 times a day if plasma bicarbonate <20 mmol/L
Lifestyle & Behavior	• Smoking cessation	Reduces CKD and CV progression
	• Physical activity	~150 min/week aerobic activity, or individualized
	• Weight control	Target BMI <25 kg/m² where feasible

Fluid & Hydration	• Maintain hydration	Routine restriction not needed until advanced CKD or volume overload
Patient Education	• Medication adherence • Recognition of warning signs (edema, dyspnea, reduced urine)	Improves engagement and early intervention
Vaccinations	• Influenza (annual) • Pneumococcal • Hepatitis B (if not immune)	Reduces risk of severe infection
Coordination of Care	• Integrate lifestyle + pharmacological strategies	Slows CKD progression, reduces CV and metabolic complications

Control of hypertension

Control BP to the target level (<130/80 mmHg) and in patients with urine albumin excretion ≥30 mg/24 hours (or equivalent), target BP is consistently <130/80 mmHg (for details, see section on Hypertension).

Tab. Amlodipine 5–20 mg/day Or Tab. Hydrochlorothiazide 12.5–25 mg Or Tab. Ramipril 5–20 mg/day Or Tab Telmisartan 40–80 mg/day. Monitor SCr and Potassium while using ACEI/ARBs. Other categories of drugs may be added if BP is not controlled.

AND/OR

Tab. Metoprolol 50–100 mg/day (Caution: contraindicated if obstructive airway disease is present)

Or

Tab. Prazosin 2.5–5 mg twice a day

Or

Tab. Clonidine 0.1 mg three times a day.

If BP is not controlled with above-mentioned measures, rule out increased salt intake and non-compliance.

Patients with severe hypertension may be advised to be admitted.

Diabetes & CKD

Managing glycaemic levels in individuals with type 2 diabetes (T2D) and CKD should involve lifestyle interventions, initial use of metformin combined with a sodium–glucose cotransporter-2 inhibitor (SGLT2i) and GLP-1 RAs as the primary treatment, and the incorporation of supplementary pharmacological measures as necessary to achieve optimal glycaemic control.

Most patients with T2D, CKD and eGFR ≥30 mL/min per 1.73 m2 would benefit from treatment with both metformin and an SGLT2i. (For details refer to guidelines on management of diabetes)

Treatment of volume overload

Many patients with CKD have volume overload especially in later stages of CKD. Salt restriction and loop diuretics like frusemide or torsemide must be prescribed to maintain volume balance.

Treatment of anaemia

1. Look for common aggravating causes of anaemia, e.g. GI blood loss, iron deficiency and chronic infections, and treat accordingly. Assess iron status of the patient before erythropoietin (EPO) therapy.
2. Provide iron supplementation to ensure adequate response to EPO. Liposomal iron has better absorption than traditional formulation. Inj. Iron Sucrose may be given (see section on Anaemia).
3. If haemoglobin is less than 10 g/dL, give Inj. Erythropoietin (EPO) 80–120 units/kg/week subcutaneous (divided into 2–3 times a week) Or long-acting erythropoiesis-stimulating agents (ESA) such as Inj. Darbepoetin alfa 0.45 mcg/kg IV (in patients undergoing dialysis) or subcutaneously once every 4 weeks or continuous erythropoietin receptor activator (CERA) once a month. These drugs have the advantage of less frequent injections and better compliance but at a higher cost.
   Note: The target Hb should be 10–12 g/dL and the optimal rate of correction should be to increase haematocrit by 4–6% over a 4-week period. To evaluate anaemia in individuals with CKD, assess haemoglobin concentration as follows: when clinically necessary for those with GFR ≥60 mL/min/1.73 m2, annually for GFR 30–59 mL/min/1.73 m2 and biannually for GFR <30 mL/min/1.73 m2.
4. Hypoxia-inducible factor prolyl hydroxylase inhibitor (HIF-PHI), also known as hypoxia-inducible factor stabilizers (HIF stabilizers), is a novel approach for managing renal anaemia. By stabilizing HIF, it stimulates erythropoiesis and increases haemoglobin levels in patients with CKD, offering a promising therapeutic strategy for addressing anaemia associated with renal dysfunction.

Treatment of bone, phosphate and calcium abnormalities and acid–base disturbances

1. Phosphate-restricted diet (avoid dry fruits, nuts and dairy-rich diet)
2. Tab. Calcium acetate/carbonate 1–2 g/day in divided doses with meals
3. Sevelamer carbonate 400–800 mg 3 times a day with meals if serum calcium levels are high
4. Must replenish vitamin D3 levels if there is deficiency of vitamin D3

5. Alfacalcidol/Calcitriol 0.25–2 mcg/day for maintenance therapy to control hyperparathyroidism
6. Tab. Sodium bicarbonate 2 g in 3–4 divided doses if there is metabolic acidosis
7. Serum calcium and phosphate maintained in normal range
8. Tab. Ferric citrate 420 mg (2 Tab. of 210 mg each) with meals 3 times a day preferred as phosphate binder in patients with normal serum calcium

Treatment of pericarditis

Uremic pericarditis is an absolute indication for initiation or intensification of dialysis. Heparin-free dialysate should be used.

Treatment of bleeding diathesis

Uremic bleeding in CKD is primarily due to platelet dysfunction. Before invasive procedures or surgery, optimize reversible factors: correct anemia (target hemoglobin appropriately), ensure adequate dialysis to reduce retained toxins, and avoid antiplatelet/anticoagulant agents if possible.

First-line pharmacologic intervention for procedure-related bleeding risk is desmopressin (DDAVP) 0.3 mcg/kg IV diluted in 50–100 mL normal saline given over 30 minutes; effect is rapid but transient (typically 4–8 hours). If bleeding recurs or risk remains high, a repeat dose can be considered cautiously, keeping in mind tachyphylaxis.

Adjuncts:

- Conjugated estrogens (e.g., oral or IV) can be used prophylactically in refractory cases, though onset is slower (days).
- Dialysis (especially shortly before procedure) helps remove uremic toxins and often improves platelet function.
- Topical hemostatic agents or local application of tranexamic acid (or systemic antifibrinolytics in selected cases) may aid in controlling localized bleeding.

Note: Routine platelet transfusion is generally not helpful unless there is concurrent true thrombocytopenia. Always review and correct any coagulopathy or contributing medication, and coordinate with nephrology and hematology for high-risk patients.

Treatment of symptomatic hyperuricaemia

Tab. Allopurinol 100–200 mg/day is given preferably after food, and then adjusted according to plasma or urinary uric acid concentration.

SUMMARY OF PHARMACOLOGICAL THERAPY IN CKD

Aspect / Drug Class	Examples	Dose / Target	Route	Key Cautions / Notes
Blood Pressure Targets	—	<140/90 mmHg (general CKD) <130/80 mmHg (if albuminuria ≥30 mg/24h)	—	Check adherence, salt intake; hospitalize if severe/uncontrolled
First-line Antihypertensives	ACE inhibitors / ARBs (Ramipril, Telmisartan, Losartan, Enalapril) CCB (Amlodipine) Thiazide diuretics (Hydrochlorothiazide*)	Ramipril 2.5–10 mg/day Telmisartan 40–80 mg/day Losartan 25–100 mg/day Enalapril 2.5–20 mg/day Amlodipine 5–10 mg/day HCTZ 12.5–25 mg/day	Oral	Monitor SCr & K^+ with ACEI/ARBs; avoid thiazides if eGFR <30 (prefer loop diuretics)
Alternative / Add-on Antihypertensives	Metoprolol, Prazosin, Clonidine	Metoprolol 50–100 mg/day Prazosin 2.5–5 mg BD Clonidine 0.1 mg TDS	Oral	Add if BP uncontrolled; avoid β-blockers in obstructive airway disease
Loop Diuretics (for volume overload)	Furosemide, Torsemide	Furosemide 20–160 mg/ day Torsemide 5–20 mg once daily (max 100 mg/ day)	Oral / IV	Monitor for dehydration, electrolyte imbalance; Torsemide has longer half-life and better oral bioavailability than furosemide
Glycemic Control (T2D + CKD)	Metformin + SGLT2i (dapagliflozin, empagliflozin, canagliflozin)	Metformin (if eGFR ≥30) + SGLT2i: dapagliflozin 10 mg OD; empagliflozin 10 mg OD; canagliflozin 100 mg OD	Oral	Stop metformin if eGFR <30 or AKI; continue SGLT2i until dialysis/ transplant unless intolerant
ESA for Anemia	Epoetin alfa, Darbepoetin alfa	Epoetin alfa 50–100 IU/kg SC weekly (individualize to Hb target)	SC / IV	Risk: hypertension, thrombosis; monitor Hb regularly
Iron Supplements	Ferrous sulfate, IV iron sucrose,	Ferrous sulfate 200 mg TDS IV iron sucrose 100–200 mg weekly ferric carboxymaltose 500–1000 mg IV per dose (not >15 mg/kg), repeat after 1 week if needed; max 1500 mg/course	Oral / IV	Correct deficiency before/with ESA; monitor ferritin & TSAT
Phosphate Binders	Metoprolol, Prazosin, Clonidine	Calcium carbonate, Calcium acetate 500–1000 mg TDS with meals; Sevelamer carbonate 800–1600 mg TDS with meals	Oral	Adjust dose to phosphate levels; monitor Ca^{2+} / phosphate balance
Vitamin D Analogues		Calcitriol 0.25–0.5 µg/day (up to 1 µg)	Oral	Monitor Ca^{2+}, phosphate; avoid hypercalcemia
Metabolic Acidosis		Sodium bicarbonate 500 mg–1 g TDS	Oral	Start if plasma bicarbonate <20 mmol/L; monitor HCO_3^- levels

IMMUNIZATION IN CKD AND DIALYSIS PATIENTS

CKD patients, especially those on dialysis or with advanced disease, are immunocompromised and at increased risk of severe infections.

Vaccine	Schedule / Dose	Special Considerations
Hepatitis B	Standard 3–4 dose series (20 µg IM at 0, 1, 6 months; or dialysis-specific double dose 40 µg at 0, 1, 2, 6 months)	Dialysis patients have reduced seroconversion—use higher dose or 4-dose schedule; check anti-HBs titers; booster if <10 mIU/mL
Influenza	Annual, inactivated quadrivalent vaccine (0.5 mL IM)	Give before influenza season each year; live attenuated vaccines not recommended in CKD
Pneumococcal	PCV20 or PCV21 (0.5 mL IM once), OR PCV15 (0.5 mL IM once) followed by PPSV23 (0.5 mL IM/SC) ≥1 year later (≥8 weeks if immunocompromised, CSF leak, cochlear implant)	Adults ≥50 years: 1 dose of conjugate vaccine (PCV20, PCV21, or PCV15+PPSV23). Adults ≥65 years: if previously only PPSV23 or older PCV, may receive PCV20 or PCV21 once (per shared decision-making). PCV20/21 alone = complete (no PPSV23 needed). Document vaccine history carefully to avoid duplication.
COVID-19	Primary series + regular boosters as per latest WHO/CDC/Ministry of Health guidance (0.5 mL IM)	CKD patients (esp. dialysis) at high risk of severe outcomes; boosters are essential. Follow latest interval recommendations.
Other vaccines (per risk & region)	• Tdap/Td (booster every 10 years) • Herpes zoster (≥50 years)	Not CKD-specific but strongly recommended in older and immunocompromised adults before influenza season each year; live attenuated vaccines not recommended in CKD

Note: Current adult pneumococcal vaccine recommendations (ACIP/CDC, 2024–25) prioritize conjugate vaccines first, then polysaccharide when indicated. Document prior vaccine history to avoid unnecessary repetition.

DIALYSIS ACCESS PLANNING (FOR DETAILS SEE ACUTE KIDNEY INJURY GUIDELINES)

Early dialysis access planning improves outcomes and reduces complications.

- Preferred access is an arteriovenous fistula (AVF); it should be created at least six months before expected dialysis starts to allow for maturation.
- Timely referral to nephrology—typically once eGFR falls below 30 mL/min/1.73 m^2—ensures evaluation, vessel mapping, and access creation can occur without delay.
- Whenever feasible, avoid initiating dialysis with temporary central venous catheters, since they carry higher risks of infection and thrombosis.

RENAL TRANSPLANTATION PLANNING

Pre-emptive transplantation (before dialysis initiation) is associated with better patient and graft survival.

- Referral to transplant centre should begin when eGFR falls below 30 mL/min/1.73 m², even if dialysis is not yet needed.
- Distinguish between:
  - Living donor transplantation: Allows for planned, pre-emptive transplantation; requires extensive evaluation of donor and recipient.
  - Deceased donor transplantation: Waitlisted patients should undergo full pre-transplant workup in advance.
- Counselling should include advantages, risks, and post-transplant care expectations.

Assessment of Response & Monitoring of CKD

- Individuals with CKD should undergo regular and stage-appropriate monitoring.
- The frequency of GFR and albumin-creatinine ratio (ACR) testing depends on CKD stage, risk factors, and the rate of disease progression.
- As CKD advances, follow-up intervals should become shorter to enable timely intervention.

Key factors influencing CKD progression include:

- Cause of CKD
- Baseline GFR and level of albuminuria
- Episodes of acute kidney injury (AKI)
- Age, sex, race/ethnicity
- Elevated blood pressure
- Hyperglycaemia
- Dyslipidaemia
- Smoking
- Obesity
- Cardiovascular disease
- Ongoing exposure to nephrotoxic agents

Note: Minor fluctuations in GFR are common and may not always indicate true disease progression.

Recommended Monitoring Schedule in CKD

Parameter	Frequency	Notes
Blood pressure, serum creatinine, electrolytes (esp. potassium), hemoglobin	**Monthly**	Essential for ongoing control and early detection of complications
eGFR, urinary albumin-to-creatinine ratio (UACR)	**Every 3 months**	Helps monitor disease progression and treatment response
Lipid profile, renal ultrasound, cardiovascular risk assessment	**Annually**	Baseline and periodic reassessment of CV risk and kidney structure

Follow-up & Treatment Adjustment by CKD Stage

CKD Stage	Recommended Follow-up Interval	Notes
Stages 1–2	Every **6–12 months**	Stable early disease; focus on risk factor control
Stage 3	Every **3–6 months**	Closer monitoring for complications, medication adjustments
Stage 4–5	Every **1–3 months**	Frequent labs and specialist review; preparation for renal replacement therapy

Issues to Address Before Step-up/Step-down

- Confirm medication adherence. Adjust medication based on renal function, electrolyte balance, and side-effect profile. Evaluate risk versus benefit of dose adjustments based on renal function.
- Address uncontrolled hypertension, diabetes, anemia, hyperkalemia, and volume status.
- Early referral to nephrologist is associated with better outcomes for patients.

Referral for Specialist Consultation

- eGFR <30 mL/min/1.73 m^2 (stage 4–5 CKD)
- Uncontrolled hypertension or diabetes
- Rapid decline in eGFR (>5 mL/min/1.73 m^2 per year)
- Refractory anemia or hyperkalemia
- Preparation for renal replacement therapy (dialysis or transplant)

RENAL REPLACEMENT THERAPY (RRT)

Modalities

- Hemodialysis (HD)
- Peritoneal dialysis (CAPD/Automated PD)
- Kidney transplantation (the only modality offering near-complete rehabilitation)

Choice of Modality

Selection depends on:

- Availability of treatment
- Patient preference and suitability
- Availability of kidney donors (for transplantation)

Patient Counselling

- RRT options should be discussed with all patients with progressive or irreversible CKD.
- Benefits and limitations of each modality must be clearly explained.
- Counseling should include preparation for vascular access and transplant referral.

Indications for Dialysis Initiation

Dialysis should begin when complications cannot be controlled medically, or at defined thresholds:

- Complications unresponsive to conservative therapy:
  - Fluid overload
  - Pericarditis
  - Refractory hyperkalemia
  - Severe metabolic acidosis
  - Uremic encephalopathy
  - Progressive uremic neuropathy

- eGFR <5 mL/min/1.73 m² irrespective of symptoms
- eGFR 5–15 mL/min/1.73 m² with symptoms of ESRD such as: unresponsive to conservative therapy:
  - Declining appetite/nutritional status
  - Persistent volume overload
  - Fatigue, malaise, mild cognitive impairment

Note: Most patients develop ESRD symptoms as eGFR falls <10 mL/min/1.73 m².

Summary of Renal Replacement Therapy (Dialysis vs Transplant)

Aspect	Hemodialysis (HD)	Peritoneal Dialysis (PD)	Kidney Transplantation
Modality	In-center or home HD using extracorporeal machine	Continuous ambulatory or automated peritoneal dialysis	Surgical placement of donor kidney
Initiation	Start when: • eGFR <5 mL/min/1.73 m² • eGFR 5–15 with symptoms (fatigue, cognitive decline, malnutrition, volume overload) • Complications unresponsive to therapy (fluid overload, refractory K^+, severe acidosis, pericarditis, encephalopathy)	Same indications as HD	Referral once eGFR <30; transplant ideally before dialysis or within 6 months of starting
Advantages	Widely available; rapid solute/fluid removal	Home-based, more flexible; preserves residual renal function longer	Best long-term survival and quality of life; near-complete rehabilitation
Limitations	Vascular access, hypotension, infections, travel burden	Peritonitis, hernia, membrane failure, requires adherence	Limited by donor availability; surgery & lifelong immunosuppression
Access / Preparation	AV fistula/graft (preferred); catheter if urgent	PD catheter placement	Early donor & recipient work-up; AV fistula preparation if likely to need interim dialysis
Complications	Hypotension, vascular access failure, infection, amyloidosis	Peritonitis, catheter infections, ultrafiltration failure	Rejection (hyperacute, acute, chronic); surgical issues (renal artery thrombosis/stenosis, urine leak, lymphocele)

KIDNEY TRANSPLANTATION

Timing of Referral

- Transplant is the **preferred therapy for ESRD**, offering best long-term survival and quality of life.
- Refer to a transplant center when eGFR <30 mL/min/1.73 m², even if dialysis has not yet begun.
- Early referral allows:
  - Recipient and donor evaluation
  - Identification of alternative donors if needed
- Best outcomes occur with pre-emptive transplant (before dialysis) or within 6 months of starting dialysis.

Pre-Transplant Evaluation

- **Medical assessment:** cardiovascular disease, infections, malignancy risk, nutritional status.
- **Social factors:** financial capacity, travel, adherence, psychosocial support.
- **Investigations:** laboratory workup, infectious disease screening, imaging, immunological profiling.

Pre-Transplant Surgical Interventions

- **Native nephrectomy/nephroureterectomy** may be required for:
  - Very large polycystic kidneys
  - Persistent heavy proteinuria
  - Reflux nephropathy with recurrent infections

Transplant Procedure and Medical Management

1. **Organ Procurement**
   - Donor identification, suitability assessment, donor maintenance.

2. **Immunosuppressive Therapy**
   - **Induction:** Basiliximab, Rabbit ATG (rATG), or Alemtuzumab.
     - High-risk of rejection → rATG
     - Low-risk → rATG or Basiliximab
   - **Maintenance- Triple therapy is standard:**
     i. Tacrolimus 0.12–0.2 mg/kg/day in 2 divided doses
     ii. Mycophenolate sodium 720 mg BD (on empty stomach) or Mycophenolate mofetil 2 g/day
     Alternative: Azathioprine 2- 2.5 mg/kg/day
     iii. Prednisolone: start 1 mg/kg/day × 3 days → taper: 20 mg/day first week → reduce 5 mg weekly until 5 mg/day maintenance
   - **Lifelong therapy required** to prevent acute and chronic rejection.
3. **Surgical/Anatomic Complications**
   - Renal artery thrombosis or stenosis
   - Urine leaks, ureteric obstruction (late)
   - Lymphocele

Allograft Dysfunction & Rejection

- Hyperacute rejection: within hours; graft loss, requires nephrectomy.
- Acute rejection: within first 6 months (~15% cases).
- Chronic rejection: >1 year; major cause of long-term graft failure.
- Other risks: drug nephrotoxicity (tacrolimus, cyclosporine), recurrence of original kidney disease.

Long-Term Follow-Up

- Regular monitoring for graft function, immunosuppressive drug levels, metabolic complications, infection, and cardiovascular risk.
- Lifelong surveillance is mandatory.

PATIENT EDUCATION

Domain	Key Education Objectives	Practical Instructions for Patients
Understanding Disease	Know CKD stage, likely trajectory, and potential complications.	• Ask your doctor about your CKD stage and what it means for your health.
Medications	Understand purpose, dosing, adherence, and avoidance of nephrotoxins.	• Take medicines exactly as prescribed. • Do NOT start new OTC drugs (esp. NSAIDs) without medical advice. • Report side effects (dizziness, low BP, urine changes). • Take phosphate binders **with meals.**
Diet & Fluids	Adhere to dietary limits for salt, protein, phosphorus, potassium; maintain hydration; correct acidosis if advised.	• Moderate salt; avoid processed foods. • Adjust protein per clinician/dietitian guidance. • Avoid high-phosphate foods (cola, dairy, processed meats). • Restrict potassium only if advised (avoid bananas, coconut water, papaya, citrus, melons if instructed). • Follow hydration advice; fluid restriction usually only in advanced stages.
Blood Pressure & Glycemic Control	Self-monitoring and strict control to slow progression.	• Check BP regularly and bring readings to appointments. • Take antihypertensives and diabetes medicines consistently. • Aim for targets set by your care team.
Lifestyle	Reduce CV risk: smoking cessation, physical activity, weight control.	• Quit smoking. • Exercise regularly (~150 min/week, or as tolerated). • Maintain healthy weight.
Vaccination & Infection Prevention	Reduce infection risk in immunocompromised patients.	• Stay updated with influenza, pneumococcal, and hepatitis B vaccines. • Follow hand hygiene and respiratory precautions.
Monitoring & Follow-up	Empower self-monitoring; recognize red flags early.	• Attend scheduled visits and labs (creatinine, electrolytes, Hb, bone profile). • Weigh yourself regularly; report sudden gains (fluid overload). • Report swelling, breathlessness, fatigue, reduced urine, confusion, or irregular heartbeat.
Preparation for Renal Replacement Therapy	Early planning for dialysis and/or transplantation.	• If advised, arrange AV fistula at least 6 months before dialysis. • Discuss HD, PD, and transplant options early. • Learn basics of transplantation, risks of rejection, need for lifelong immunosuppression, and monitoring.
Self-care & Communication	Encourage shared decision-making, caregiver involvement, and advance care planning.	• Keep a list of all medicines and share with all providers. • Involve family or caregivers in your care discussions. • Ask questions when unclear. • Discuss future care preferences (advance care planning).

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