Kidney Health® Australia

5th Edition

Chronic Kidney Disease (CKD) Management in Primary Care

Guidance and clinical tips to help detect, manage, and refer patients with CKD in your practice

ANZSN

apna Supporting nurses in primary health care

RSA Renal Society of Australasia

RACGP Accepted clinical resource

Evidence review

The recommendations in the *'Chronic Kidney Disease (CKD) Management in Primary Care'* handbook are based on consideration of current evidence, Australian and international guidelines and clinical consensus. The goal of the handbook is to provide simple and actionable advice for health professionals on the detection and management of CKD.

Endorsements

The *'Chronic Kidney Disease (CKD) Management in Primary Care'* (5th edition) handbook has been officially recognised as an Accepted Clinical Resource by The Royal Australian College of General Practitioners (RACGP) and endorsed by the Australia and New Zealand Society of Nephrology (ANZSN), Primary Healthcare Nurses Association (APNA) and the Renal Society of Australasia (RSA).

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Disclaimer

The recommendations contained in this handbook were formed from existing evidence-based clinical guidelines, current research, and clinical consensus. The guidance is based upon the best information available at the time of publication. It is designed to provide information and assist decision-making. It is not intended to indicate an exclusive course of action or serve as a standard of medical care. Variations, taking individual circumstances into account, may be appropriate. Every health-care professional making use of this guide is responsible for evaluating the appropriateness of applying it in the setting of any particular clinical situation. The authors assume no responsibility for personal or other injury, loss or damage that may result from the information in this publication. Please note that requirements for PBS subsidy may differ from recommendations contained in this guide.

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PEAK Committee

Prof David Johnson (chair), Nephrologist
Prof Karen Dwyer, Nephrologist
A/Prof Shilpa Jesudason, Nephrologist
A/Prof Ivor Katz, Nephrologist
A/Prof Craig Nelson, Nephrologist
Dr Richard Phoon, Nephrologist
Prof Kevan Polkinghorne, Nephrologist
Dr Angus Ritchie, Nephrologist
Dr John Saunders, Nephrologist
Dr Chris Bollen, General Practitioner
Dr Caitlin Sum, General Practitioner
Prof Tim Usherwood, General Practitioner
Dr Sheena Wilmot, General Practitioner
Dr Leanne Brown, Renal Nurse Practitioner
Ms Jo Lewis, Nurse Practitioner
Mr Tim Perry, Pharmacist

Kidney Health Australia

Breonny Robson
Katrin Buerkle
Claire Sheeky

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We acknowledge the Traditional Owners of Country throughout Australia and recognise their continuing connection to lands, waters, and communities. We pay our respect to Aboriginal and Torres Strait Islander cultures, and to Elders both past and present.

Conflicts of interest: DJ has received consultancy fees, research grants, speaker's honoraria and travel sponsorships from Baxter Healthcare and Fresenius Medical Care (FMC), consultancy fees from AstraZeneca (AZ), Bayer, and AWAK technologies, speaker's honoraria from Ono pharmaceutical and Boehringer Ingelheim (BI) & Lilly, and travel sponsorships from Ono pharmaceutical and Amgen. He is a current recipient of an Australian NHMRC Leadership Investigator Grant. CB is a director of BMP Healthcare Consulting (BMP), which has been engaged by Kidney Health Australia (KHA) in the delivery of educational activities. BMP also hold contracts with multiple PHNs across Australia to deliver programs. KD has received honorarium from AZ, BI, Bayer, GSK. She has received seed grant funding from Servier; Western Alliance, and Deakin University, as well as funding through NHMRC, MRFF, Deakin University, Epworth Medical Foundation. She was the KHA Clinical Director from 2021-23. SJ was the KHA Clinical Director from 2017 to 2020. RP received consultancy fees and honoraria from AZ. AM is the recipient of a Qld Health Advancing Clinical Research Fellowship, has received research funding (competitive, investigator-initiated) from Sanofi-Genzyme, NHMRC, MRFF, QLD Health, and Australian Government Department of Health, as well as travel support from Otsuka. He also holds advisory board membership with GSK and MSD (no honoraria, travel support or payments), and board membership with ANZSN (Council). CS received speakers' fees from AZ and Astellas. BR has taken part in advisory boards for AZ and GSK. All other contributors have no conflicts of interest to declare.

Foreword

Welcome to the 5th edition of the *'Chronic Kidney Disease (CKD) Management in Primary Care'* handbook. This new edition provides clear, succinct, up to date, best-practice guidance for the care of patients at risk of or living with CKD, including identification of those at risk, early detection of CKD, lifestyle and pharmacologic management, reduction of kidney and cardiovascular risks, and quality use of medicines in the setting of CKD.

CKD is a growing public health problem in Australia that is common, harmful, and treatable. More than 6 million Australian adults are at risk of CKD and more than 2 million live with CKD, including over 31,000 people with kidney failure requiring dialysis or kidney transplantation. People with early-stage CKD are up to 20 times more likely to die from premature cardiovascular disease than survive to the point of needing kidney replacement therapy. In 2021, it was estimated that CKD caused the loss of over 55,000 healthy life years and cost the Australian community $9.9 billion dollars per annum. CKD is silent until as much as 90% of kidney function is lost and the vast majority (94%) of those with kidney disease are unaware that they have the condition. This means that CKD is generally not detected until it reaches a more serious, burdensome, and costly stage.

Primary care clinicians play a pivotal role in the early detection and care of people with CKD. This role has become even more critical with the recent emergence of new, highly effective treatments. The extensively used *'CKD Management in Primary Care'* handbook, and its accompanying freely downloadable digital application, CKD-Go!, have been comprehensively updated with the latest, best-practice recommendations, including new treatments (e.g. SGLT-2 inhibitors and non-steroidal mineralocorticoid receptor antagonists), new management algorithms, new information on how the updated Aus CVD risk calculator should be applied to CKD, new information on lifestyle approaches, and new recommendations on providing culturally safe care to First Nations Australians living with CKD.

The handbook is the product of Herculean efforts made by members of the Primary Care Education Advisory Committee to Kidney Health Australia (PEAK) and the amazing Primary Care Education Team at Kidney Health Australia. We gratefully acknowledge their enormous contributions and, in particular, would like to sincerely thank outgoing longstanding members, Professor Tim Usherwood and Professor Robyn Langham, who have tirelessly provided outstanding inputs over the last two decades.

We hope that this latest edition of the *'Chronic Kidney Disease (CKD) Management in Primary Care'* handbook will be both an invaluable and indispensable guide to primary healthcare professionals providing care to people with CKD in the community.

Prof David Johnson
Chair of PEAK Committee
Kidney Health Australia

Why use this handbook?

The *'Chronic Kidney Disease (CKD) Management in Primary Care'* (5th edition) handbook provides best practice recommendations for detecting and managing CKD in primary care:

- Easy to use and interactive.
- Advice on detecting CKD in primary care including required tests and easy to use algorithm.
- Colour-coded CKD staging table.
- Colour-coded clinical action plans outlining goals of management, key management tasks and treatments to slow the progression of CKD.
- Medication advice and treatment targets.
- Management framework for common issues in CKD.
- Nephrology referral algorithm.
- Links to fact sheets, websites, and additional resources for you and your patients.

The *'CKD Management in Primary Care'* handbook is available in both hardcopy and electronic copy at kidney.org.au. It is also available via the app 'CKD-Go!' (Free download from your app store).

What's new in the 5th edition?

- New recommendations for the use of medications to slow progression of CKD and reduce cardiovascular (CVD) risk.
- Revised algorithm for the initial detection and diagnosis of CKD.
- New guidelines for culturally safe kidney care in First Nations Australians.
- New information on the updated Aus CVD risk calculator and how it applies to patients at varying stages of CKD.
- Expanded information on managing nutrition and diet in CKD.
- New guidelines and algorithm for referral of people with CKD to kidney specialists.
- New recommendations on a stepwise approach to albuminuria reduction in CKD.
- New sections on genetic kidney disease, pharmacological management, heart failure and CKD, pain management, contraception and pregnancy, and hyperuricaemia.
- Links to additional information, resources, and apps, including the *'Kidney Health Australia Health Professional Hub'*.

"Early diagnosis means people like me can control their lifestyle and can make a difference in society for longer, rather than being reliant on society."

Shailendra
Kidney Advocate living with CKD

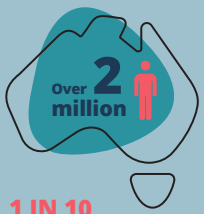
Contents

CKD in Australia

Common

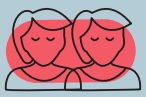
CKD IS **TWICE AS COMMON** AS DIABETES.[1]

3 OUT OF 4 AUSTRALIAN ADULTS HAVE AT LEAST ONE FACTOR INCREASING THEIR RISK OF CKD.[2]



1 IN 10 AUSTRALIANS AND **1 IN 5** FIRST NATIONS AUSTRALIANS AGED 18 YEARS AND OVER ARE LIVING WITH SIGNS OF CKD.[1]

Harmful



THE NUMBER OF PATIENTS NEEDING TREATMENT FOR KIDNEY FAILURE HAS **DOUBLED** IN THE LAST 20 YEARS.[2]

20x

PEOPLE WITH CKD ARE UP TO **20 TIMES** MORE LIKELY TO **DIE FROM A HEART ATTACK** OR STROKE THAN THEY ARE TO PROGRESS TO KIDNEY FAILURE.[3-5]

THE **BURDEN OF CKD** IS GREATEST IN PEOPLE EXPERIENCING SOCIOECONOMIC DISADVANTAGE, LIVING RURALLY AND IN FIRST NATIONS AUSTRALIANS.[2]

AROUND **20,000** AUSTRALIANS **DIE EVERY YEAR** WITH KIDNEY DISEASE.[2]

CKD CAN HAVE **SIGNIFICANT IMPACT** ON WORK, FAMILY, AND PSYCHOSOCIAL **WELLBEING**.[6-11]

CKD CONTRIBUTES TO **1 IN 6 HOSPITALISATIONS** IN AUSTRALIA.[2]

Treatable

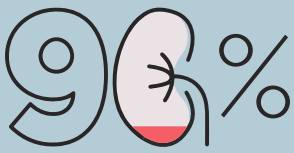
NEW TREATMENTS CAN SLOW THE PROGRESSION OF CKD BY UP TO **15 YEARS**,[13] OR POTENTIALLY LONGER IF STARTED EARLY.[14]

IF **CKD IS DETECTED EARLY** AND MANAGED APPROPRIATELY, DETERIORATION IN KIDNEY FUNCTION CAN BE **REDUCED BY AS MUCH AS 50%**.[12]

Often overlooked

FEWER THAN 10% OF THE PEOPLE WITH CKD ARE AWARE THAT THEY HAVE THIS CONDITION.[1]

LATE REFERRAL IS COMMON. 17% OF PEOPLE COMMENCE DIALYSIS WITHIN 90 DAYS OF BEING REFERRED TO A KIDNEY SERVICE.[15]



90% OF KIDNEY FUNCTION CAN BE LOST BEFORE PEOPLE EXPERIENCE SYMPTOMS.

Treatment goals for people with CKD

Parameter	Treatment goal	
Nutrition and diet	• Adopt a healthy dietary pattern that includes vegetables, fruit, wholegrains, nuts and legumes, dairy foods, lean meat, fish, and plant protein. • Reduce salt intake to <5 g per day. • Avoid ultra-processed foods and sugar-sweetened drinks. • Drink water to satisfy thirst.	• See page 30
Weight management	• BMI: <25 kg/m^2 (<23 kg/m^2 Asian population[16]). • Waist circumference <94 cm in men (<90 cm in Asian men) or <80 cm in women (including Asian women).	• See page 32
Physical activity	• Be active on most days, preferably every day. • Aim for 2.5 – 5 hours of moderate intensity activity across the week[17], or at a level that accounts for the individual's cardiovascular and physical health.[18] • Some activity is better than none. • Include muscle strengthening activities as part of daily activity at least twice per week.[17]	• Physical activity and exercise guidelines for all Australians
Smoking/ Vaping	• Don't smoke or vape. To aid smoking/vaping cessation, recommend counselling and if required, nicotine replacement therapy or other medication.	• Quitline 13 7848
Alcohol	• Reduce alcohol consumption. The less you drink, the lower your risk of harm from alcohol.[19] • Australian guidelines for 'healthy' individuals recommend no more than 10 standard drinks a week and no more than 4 standard drinks on any one day to reduce the risk of harm from alcohol-related disease or injury.[19] • There are no specific recommendations about safe levels of alcohol consumption in people with CKD.	• Australian guidelines to reduce health risks from drinking alcohol

Hypertension	• Maintain blood pressure consistently below 130/80 mmHg for all people with CKD.	• See page 52
Glycaemic control	• Maintain blood glucose levels (BGL): 6-8 mmol/L fasting; 8-10 mmol/L postprandial. • HbA1c: ≤53 mmol/mol (range 48-58); ≤7% (range 6.5-7.5). Individualise according to patients' circumstances (e.g., disease duration, life expectancy, important comorbidities, and established vascular complications).	• See page 49 • Management of type 2 diabetes: A handbook for general practice • Diabetes Australia
Albuminuria	• A reduction in uACR of at least 30%.[20-23]	• See page 59
Lipids	• No target serum cholesterol level recommended. • Use statin (+/- ezetimibe) to reduce risk of CVD events and death in: – People with CKD (eGFR ≥15 mL/min/1.73m^2) and CVD risk ≥10%. – First Nations Australians with CKD and CVD risk ≥5%).	• CARI guidelines: Management of lipid-lowering therapy in CKD • Aus CVD risk calculator
Potassium	• K^+ ≤6.0 mmol/L.	• See page 65
Immunisation	• Recommended vaccinations for people with CKD aged 18 years and over: – Influenza – Pneumococcal – COVID-19 – Herpes Zoster* – Other vaccinations as recommended by the NIP	• National Immunisation Program • The Australian Immunisation Handbook

* Herpes Zoster vaccinations are currently on the National Immunisation Program (NIP) for all people aged 65 years and over; First Nations peoples aged 50 years and over; and immunocompromised persons aged 18 years and over who have had a kidney transplant or another eligible medical condition.

Please note that some recommendations contained in this handbook may differ to current Pharmaceutical Benefits Scheme (PBS) subsidy indications.

We recommend checking the PBS and NIP listings for specifics, before prescribing.

1

Detecting and diagnosing CKD

Clinical presentation of CKD

CKD is defined as:

An estimated or measured GFR <60 mL/min/1.73m^2 that is present for ≥3 months with or without evidence of kidney damage.

Or

Evidence of kidney damage, with or without decreased GFR that is present for ≥3 months, as evidenced by the following:

- Albuminuria
- Haematuria after exclusion of urological causes
- Structural abnormalities (e.g., on kidney imaging tests)
- Pathological abnormalities (e.g., kidney biopsy)

CKD is generally asymptomatic

- Up to 90% of kidney function may be lost before symptoms are present, so 1-2 yearly checks for individuals at increased risk are essential.

Signs and symptoms of advancing CKD may be general in nature and include:

- Hypertension
- Pruritis
- Nocturia
- Lethargy
- Nausea/vomiting
- Malaise
- Anorexia
- Restless legs
- Haematuria
- Dyspnoea

Targeted detection of CKD

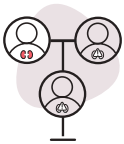
Three out of four Australians aged 18 years and over are at increased risk of developing CKD. Detection of CKD should be targeted and focus on people who are known to be at increased risk of developing the condition.

The following people are at increased risk of CKD:

Diabetes

Hypertension

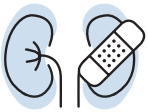
Established cardiovascular disease



Family history of kidney failure

Obesity (body mass index ≥30kg/m²

Current or past smoker/vaper



History of acute kidney injury

All people aged 60 years or older

First Nations Australians aged 18 years or older*

* First Nations Australians experience a higher burden, earlier onset, and faster progression of kidney disease, due to ongoing impacts of colonisation.

Clinical tip

Individuals at increased risk of developing CKD should be offered a Kidney Health Check (BP, uACR, eGFR) every 1-2 years (annually for First Nations Australians aged ≥18 years, and anyone with diabetes or hypertension).

See page 13 for information on testing people at risk of CKD.

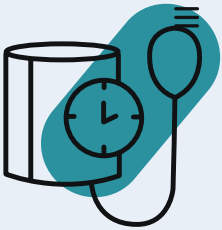
Early detection of CKD

Benefits of early detection

- Allows for early intervention with treatment approaches and medications that can slow the progression of kidney disease and reduce cardiovascular risk.
- Facilitates appropriate adjustments to drug dosing, prescribing of medications to slow CKD progression, establishment of a sick day action plan, and avoidance of nephrotoxic medications.
- Gives patients time to make positive lifestyle changes and appropriately consider treatment options before reaching kidney failure.
- Has economic benefits for the health system, with every $1 invested leading to $45 of savings in the cost of treating kidney failure and associated CVD.[24]

The Kidney Health Check

Targeted detection of CKD occurs via Kidney Health Checks for patients at increased risk. The Kidney Health Check involves the following:



Blood pressure check
Consider ambulatory blood pressure (ABP) monitoring or home BP monitoring in addition to in-clinic readings.

Urine test
Albumin/creatinine ratio (ACR) to check for albuminuria.

Blood test
eGFR calculated from serum creatinine.

Further information and resources

- Kidney Health Australia Health Professional Hub: How to guide - How to Kidney Health Check with MBS item numbers.
- Kidney Health Australia factsheet for people affected by CKD: What is CKD?

Early detection of CKD in non-Indigenous Australians

Indications for assessment[#^]	Recommended frequency	Assessment
Diabetes	Annually	Complete a **Kidney Health Check**: • Blood pressure check • uACR (first morning void preferred) • eGFR If results indicate CKD, repeat tests, refer to the algorithm for initial detection and diagnosis of CKD on page 16.
Hypertension	Annually	
Established cardiovascular disease[*]	Every 2 years	
Family history of kidney failure	Every 2 years	
Obesity	Every 2 years	
Smoking / vaping	Every 2 years	
History of acute kidney injury (AKI)	Every year for first 3 years post AKI, then every 2 years.	
Aged ≥60 years	Once off, unless developing other indications for assessment.	

* Established cardiovascular disease is defined as a previous diagnosis of coronary heart disease, cerebrovascular or peripheral vascular disease.

\# There is emerging evidence that the long-term effects of COVID-19 may also increase the risk of future kidney disease.[25] Therefore, a Kidney Health Check is recommended for individuals who have experienced acute kidney injury (AKI) as a complication of COVID-19 infection and for those with long COVID.

^ A Kidney Health Check is recommended for people who have had hypertensive disorders in pregnancy, including pre-eclampsia and pregnancy-induced hypertension, which are associated with increased future risk of hypertension, cardiovascular disease, and CKD.

Early detection of CKD in First Nations Australians[26]

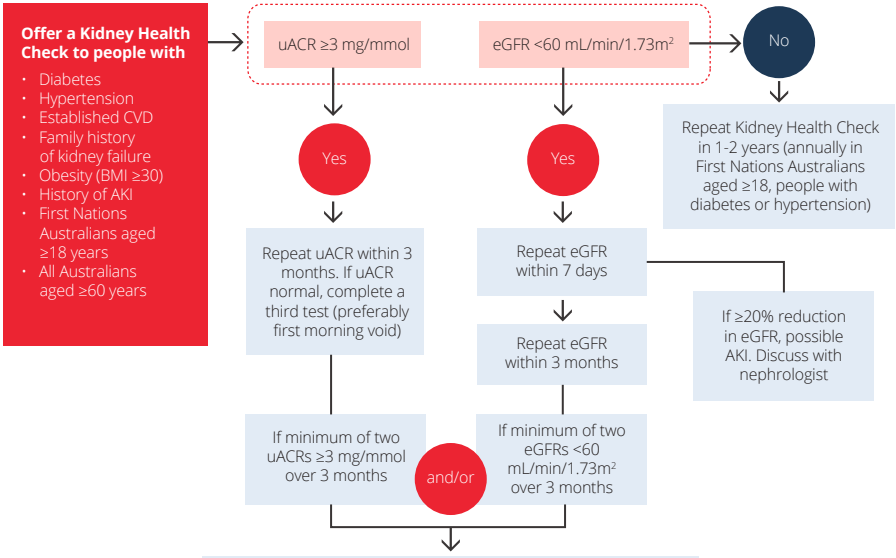
Indications for assessment#^*	Recommended frequency	Assessment
Aged <18 years	As needed	Screen for 'red flags' of CKD: • Family history of CKD • Clinical history of diabetes, hypertension, obesity, smoking, established CVD, or acute kidney injury • Clinical history of low birth weight • Clinical history of recurrent childhood infections Also consider: • Socioeconomic status, regional/rural and remote location, housing status, education level Complete a Kidney Health Check (eGFR, uACR and blood pressure check) if concerned.
Aged ≥18 years	At least annually. Utilise MBS item 715 if appropriate.	Complete a **Kidney Health Check** • Blood pressure check • uACR (first morning void preferred) • eGFR If results indicate CKD, repeat tests, refer to the algorithm for initial detection and diagnosis of CKD on page 16.

#^* See footnotes on the definition of established cardiovascular disease, and testing recommendations for people with AKI associated with COVID-19, long COVID and hypertensive pregnancy disorders on page 14.

Further information and resources

- CARI Guidelines: CARI Recommendations for culturally safe kidney care for First Nations Australians.
- RACGP Red Book: Guidelines for preventive activities in general practice.
- Kidney Health Australia education tool for health professionals: Flipchart for First Nations Australians.
- Kidney Health Australia resource library: Factsheets for First Nations Australians affected by CKD.
- Kidney Health Australia's 'CKD–Go!' app available on your app store. The 'CKD–Go!' app has an inbuilt CKD calculator to quickly determine a person's clinical management plan.

Algorithm for initial detection and diagnosis of CKD



		Albuminuria Stage		
Kidney Function Stage	**GFR (mL/min/1.73m²)**	**Normal (A1) uACR <3.0 mg/mmol**	**Microalbuminuria (A2) uACR 3.0-30 mg/mmol**	**Macroalbuminuria (A3) uACR >30 mg/mmol**
1	≥90	Not CKD unless haematuria, structural or pathological abnormalities present		
2	60-89			
3a	45-59			
3b	30-44			
4	15-29			
5	<15 or on dialysis			

Undertake investigations to determine underlying diagnosis

Fully specify CKD diagnosis, e.g CKD stage 2 with microalbuminuria (A2) in the presence of type 2 diabetes

Refer to the colour-coded clinical action plans for CKD management strategies:

Yellow clinical action plan (p26) **Orange clinical action plan (p27)** **Red clinical action plan (p28)**

Tests used to investigate CKD

Glomerular filtration rate (GFR)[27]

- GFR is accepted as the best overall measure of kidney function.
- GFR can be estimated (eGFR) from serum creatinine using the CKD-EPI prediction equation and is routinely reported by all Australian pathology laboratories with requests for serum creatinine in individuals aged ≥18 years.
- Use of the CKD-EPI equation has been validated in First Nations Australians, as well as South-East Asian, African, Indian, and Chinese people living in western countries. No adjustments to the equation are needed to measure eGFR in different population groups.[27]
- eGFR is a more sensitive marker for CKD than serum creatinine alone.
- Normal serum creatinine measurements do not exclude serious loss of kidney function.
- 50% or more of kidney function can be lost before the serum creatinine rises above the upper limit of normal.
- In the context of normal uACR results, further investigation of reduced eGFR is usually only required if the eGFR is <60 mL/min/1.73m^2 or declining.

Clinical situations where eGFR results may be unreliable and/or misleading:[28]

- Acute changes in kidney function (e.g., AKI).
- People on dialysis.
- Recent consumption of cooked meat (consider re-assessment when the individual has fasted or specifically avoided a cooked meat meal within 4 hours of blood sampling).
- Exceptional dietary intake (e.g., vegetarian diet, high protein diet, creatine supplements).
- Extremes of body size.
- Conditions of skeletal muscle, paraplegia, or amputees (may overestimate eGFR).
- High muscle mass (may underestimate eGFR).
- People under the age of 18 years.
- Severe liver disease present.
- eGFR values above 90 mL/min/1.73m^2.
- Drugs interacting with creatinine excretion (e.g., trimethoprim).
- Pregnancy (see below).
- Minor changes in eGFR (≤15% change) could be due to physiological or laboratory variability.

eGFR and drug dosing[27]

- Dose reduction of some drugs is recommended for people with reduced kidney function (see page 34).
- eGFR provides a valid estimate of kidney drug clearance and is widely available on laboratory reports.
- If using eGFR for drug dosing, body size should be considered, in addition to referring to the approved product information.
- For drug dosing in very large or very small people, it may be preferred to calculate an eGFR that is not normalised to 1.73m^2 body surface area (BSA).
- For drugs with a narrow therapeutic index, therapeutic drug monitoring or a valid marker of drug effect should be used to individualise dosing.

eGFR and pregnancy

- The validity of eGFR in pregnancy is not known.
- The use of eGFR to assess kidney function in pregnant people is not recommended.
- Serum creatinine should remain the standard test for kidney function in pregnant people.

Clinical tip

If eGFR is <60 mL/min/1.73m^2, retest within 7 days to exclude AKI and again after 3 months to establish a diagnosis of CKD. Also consider clinical situations where eGFR results may be unreliable and/or misleading.

Urine albumin/creatinine ratio (uACR)

- Protein in the urine is a key marker of kidney damage and linked to increased risk of progression to kidney failure and cardiovascular disease.
- These proteins are mainly albumin (albuminuria), but also consist of low molecular weight immunoglobulin, lysozyme, insulin, and beta-2 microglobulin.
- It is rare for an individual to have increased excretion of non-albumin proteins without concomitant increased excretion of albumin.
- An increased uACR is predictive of heightened kidney and cardiovascular risks in population studies.
- Reduction in uACR confers reno-protective benefit in intervention trials.[29]
- Elevated uACR is a more common sign of CKD than a decreased eGFR and is commonly missed as part of a Kidney Health Check in practice.[1]

How to detect albuminuria:[30]

- Urinary protein excretion follows a circadian pattern and tends to be highest in the afternoon, so uACR tests are most accurate when performed in the early morning (first void), and this is the preferred method for assessment of albuminuria.[31]
- Where a first void specimen is not possible or practical, a random spot urine specimen for uACR is acceptable.
- A positive uACR test (uACR ≥3.0 mg/mmol) should be repeated on a first void sample to confirm persistence of albuminuria.
- Dipstick for protein in the urine is no longer recommended, due to poor sensitivity and specificity.
- uACR exhibits greater sensitivity than urine protein/creatinine ratio (uPCR) for detecting lower amounts of clinically important albuminuria. uPCR tests may miss microalbuminuria, resulting in false-negative results.
- In general, 24-hour urine collection is not warranted to quantify proteinuria.
- uACR criteria for CKD is not applicable in pregnancy.

Factors other than CKD known to increase urine albumin excretion:[30]

- Urinary tract infection
- High dietary protein intake
- Congestive heart failure
- Acute febrile illness
- Heavy exercise within 24-hours
- Menstruation
- Genital discharge or infection
- Drugs (especially NSAIDs)

Clinical tip

Albuminuria is present, if at least two uACR results are ≥3.0 mg/mmol. If this is consistent over a 3-month period, it is indicative of CKD.

Other diagnostic evaluation tests for CKD:[32]

Always indicated:

- Repeat (within 1 week) serum urea/electrolytes/creatinine/eGFR/albumin tests.
- If eGFR continues to decrease, refer to AKI management plan (see page 40).
- Urine microscopy for dysmorphic red cells, red cell casts
- Full blood count, fasting lipids, glucose, HbA1c, uric acid, LFTs, hsCRP, ESR
- Ultrasound of the kidneys, ureters, and bladder (KUB) (at least once)

Sometimes indicated:

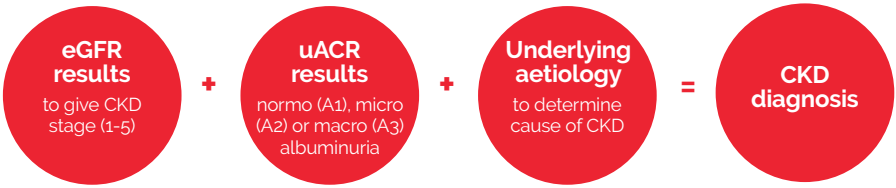
If the following is present:	Carry out the following test:
Signs of systemic disease (e.g., rash, arthritis, features of connective tissue disease, pulmonary symptoms or deteriorating kidney function) or the presence of glomerular haematuria. Prompt referral likely indicated.	• Anti-glomerular basement membrane antibody (antiGBM) • Anti-neutrophil cytoplasmic antibody (ANCA) • Anti-nuclear antibodies (ANA) • Extractable nuclear antigens (ENA) • Complement studies (C3/C4) • ESR
Risk factors for HBV, HCV, or HIV (these conditions are associated with an increased risk of glomerular disease).	HBV, HCV, HIV serology
Possible myeloma.	Serum, urine protein electrophoresis and FLC

Further information and resources

- Kidney Health Australia factsheets for people affected by CKD: 'What is eGFR?' and 'Albuminuria'.
- Kidney Health Australia Health Professional Hub: How to guide - Stage & Diagnose CKD.

Diagnosing CKD

There are three components to a diagnosis of CKD



Clinical tip

CKD in itself is not a primary diagnosis. Attempts should be made to identify the underlying cause of CKD and to fully specify it, e.g., CKD stage 3b with microalbuminuria (or A2) in the presence of type 2 diabetes. Remember to code all CKD diagnoses in your practice software.

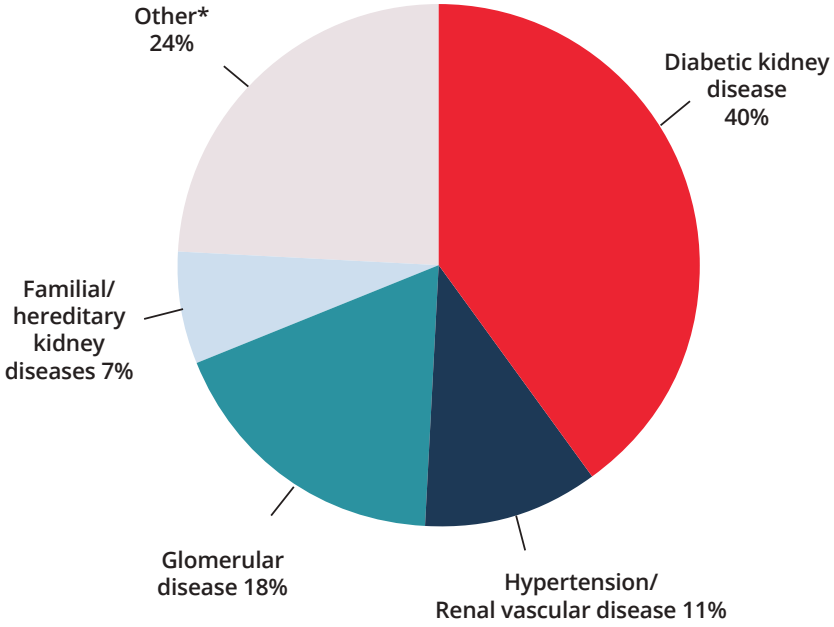
Staging of CKD

Once an individual is diagnosed with CKD, it is useful to determine their CKD stage and corresponding CKD action plan as this will guide management strategies.

To correctly stage CKD, combine the person's kidney function stage (stage 1-5 determined by their eGFR) with their albuminuria stage. This is then plotted on the colour-coded CKD staging table to determine which action plan they should follow. The staging table colours are indicative of increasing risk of kidney failure and CVD, with green indicating minimal risk and red indicating highest risk.

Causes of kidney failure

The most common causes of kidney failure in Australia are:[15]



* 'Other' includes: tubulointerstitial disease (8%), other systemic diseases affecting the kidney (2%), miscellaneous kidney disorders (13%), not reported (1%).

Genetic kidney diseases

An important cause of CKD is genetic kidney disease. Whilst overall this is a minority of cases, it remains significant and includes conditions such as autosomal dominant polycystic kidney disease (ADPKD) and Alport syndrome. Key factors to consider include:

- Family history
- Age of onset (particularly in childhood)
- Extra-kidney manifestations
- Genetic testing in people aged 50 years or younger without known cause

In addition, there can be a variety of pathognomonic features for many individual disorders or syndromes. The inclusion of the above information and any specific concerns for a heritable or genetic form of kidney disease can be communicated at the time of nephrology referral.

Further information and resources

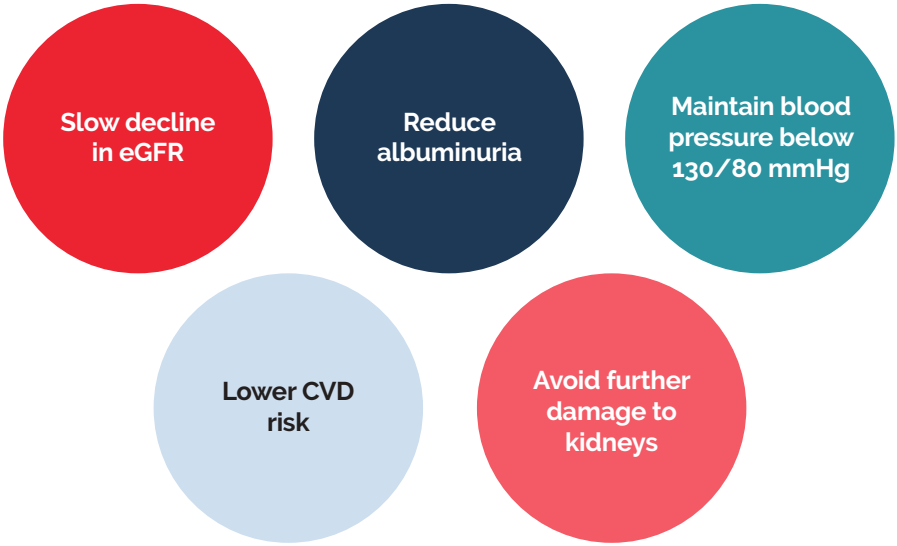
A range of support organisations are available for those with a diagnosis of genetic kidney disease, including:

- PKD Australia.
- Alport Foundation of Australia.
- KidGen Collaborative.
- CARI Guidelines: Autosomal Dominant Polycystic Kidney Disease.

2

Managing CKD in Primary Care

Key management strategies for all people with CKD



Clinical tip

Following diagnosis and staging of CKD, use the corresponding colour-coded clinical action plan for key management strategies key management goals and strategies..

Yellow clinical action plan

eGFR ≥60mL/min/1.73m² with microalbuminuria (A2) or
eGFR 45-59 mL/min/1.73m² with normoalbuminuria (A1)

Management goals

- Slow progression of CKD.
  - Slow decline in eGFR.
  - Reduce albuminuria by at least 30%.
- Assess and lower cardiovascular risk.
- Avoid nephrotoxic medications or volume depletion.
- Encourage positive lifestyle changes and self-management practices.

Management strategies

Frequency of review
- Every 12 months

Clinical assessment
- Blood pressure
- Weight and waist circumference
- Smoking/vaping history

Laboratory assessment

Recommended:
- uACR (see page 18)
- eGFR (see page 17)
- Urea, creatinine, and electrolytes
- Full blood count

Also consider:
- Screening for diabetes (fasting blood glucose or HbA1c)
- HbA1c (for people with diabetes)
- Dipstick urinalysis for haematuria detection
- Lipid studies (Trig, Chol, HDLC, LDLC)

Treatment checklist
- Complete investigations to determine underlying cause of CKD.
- Provide advice on positive lifestyle changes (addressing smoking/vaping, nutrition, alcohol use, physical activity, sleep, stress) (see page 29-32).
- Maintain blood pressure consistently below target (see page 52).
- Complete cardiovascular risk assessment (see page 48).
- Prescribe medications to slow CKD progression, e.g., ACE inhibitor or ARB, SGLT2 inhibitor, non-steroidal MRA (see page 34).
- Consider lipid lowering treatment where appropriate (see page 65).
- Optimise glycaemic control (see page 49).
- Avoid nephrotoxic medications or volume depletion (see page 35).
- Discuss contraception with individuals of child-bearing age (see page 37).
- Recommend vaccinations (see page 39).

Orange clinical action plan

eGFR 30-59mL/min/1.73m² with microalbuminuria (A2) or
eGFR 30-44 mL/min/1.73m² with normoalbuminuria (A1)

Management goals

- Slow progression of CKD.
  - Slow decline in eGFR.
  - Reduce albuminuria by at least 30%.
- Assess and lower cardiovascular risk.
- Avoid nephrotoxic medications or volume depletion.
- Encourage positive lifestyle changes and self-management practices.

+

- Early detection and management of complications.
- Adjust medication doses to levels appropriate for kidney function.
- Appropriate referral to a nephrologist when indicated.

+

Management strategies

Frequency of review
- Every 3-6 months

Clinical assessment
- Blood pressure
- Weight and waist circumference
- Smoking/vaping history

Laboratory assessment

Recommended:
- uACR (see page 18)
- eGFR (see page 17)
- Urea, creatinine, and electrolytes
- Full blood count

Also consider:
- Screening for diabetes (fasting blood glucose or HbA1c)
- HbA1c (for people with diabetes)
- Dipstick urinalysis for haematuria detection
- Lipid studies (Trig, Chol, HDLC, LDLC)
- Iron studies
- Calcium and phosphate
- Parathyroid hormone (6-12 monthly if eGFR <45mL/min/1.73m²)

Treatment checklist
- Complete investigations to determine underlying cause of CKD.
- Provide advice on positive lifestyle changes (addressing smoking/vaping, nutrition, alcohol use, physical activity, sleep, stress) (see page 29-32).
- Maintain blood pressure consistently below target (see page 52).
- Complete cardiovascular risk assessment (see page 48).
- Prescribe medications to slow CKD progression, e.g., ACE inhibitor or ARB, SGLT2 inhibitor, non-steroidal MRA (see page 34).
- Consider lipid lowering treatment where appropriate (see page 65).
- Optimise glycaemic control (see page 49).
- Avoid nephrotoxic medication or volume depletion and adjust doses to levels appropriate for kidney function (see page 35).
- Assess for common issues presenting in CKD (see pages 58-71).
- Appropriate referral to nephrologist when indicated (see page 73).
- Discuss contraception with individuals of child-bearing age (see page 37).
- Recommend vaccinations (see page 39).

Red clinical action plan

Macroalbuminuria irrespective of eGFR or
eGFR <30 mL/min/1.73m² irrespective of albuminuria

Management goals

- Slow progression of CKD.
  - Slow decline in eGFR.
  - Reduce albuminuria by at least 30%.
- Assess and lower cardiovascular risk.
- Avoid nephrotoxic medications or volume depletion.
- Encourage positive lifestyle changes and self-management practices.

+

- Early detection and management of complications.
- Adjust medication doses to levels appropriate for kidney function.
- Appropriate referral to a nephrologist when indicated.

+

- Prepare for kidney replacement therapy if appropriate.
- Prepare for comprehensive conservative care if appropriate.

Management strategies

Frequency of review
- Every 1-3 months

Clinical assessment
- Blood pressure
- Weight and waist circumference
- Smoking/vaping history
- Oedema

Laboratory assessment

Recommended:
- uACR (see page 18)
- eGFR (see page 17)
- Urea, creatinine, and electrolytes
- Full blood count

Also consider:
- Screening for diabetes (fasting blood glucose or HbA1c)
- HbA1c (for people with diabetes)
- Dipstick urinalysis for haematuria detection
- Lipid studies (Trig, Chol, HDLC, LDLC)
- Iron studies
- Calcium and phosphate
- Parathyroid hormone (6-12 monthly if eGFR <45mL/min/1.73m²)

Treatment checklist
- Complete investigations to determine underlying cause of CKD.
- Provide advice on positive lifestyle changes (addressing smoking/vaping, nutrition, alcohol use, physical activity, sleep, stress) (see page 29-32).
- Maintain blood pressure consistently below target (see page 52).
- Address high cardiovascular risk (see page 48).
- Prescribe medications to slow CKD progression as relevant to the persons eGFR, e.g., ACE inhibitor or ARB, SGLT2 inhibitor, non-steroidal MRA (see page 34).*
- Consider lipid lowering treatment where appropriate (see page 65).
- Optimise glycaemic control (see page 49).
- Avoid nephrotoxic medication or volume depletion and adjust doses to levels appropriate for kidney function (see page 35).
- Assess for common issues presenting in CKD (see pages 59-71).
- Appropriate referral to nephrologist when indicated (see page 74).
- Discuss potential progression to kidney failure with patient and treatments.
- Initiate advance care planning (see page 75)
- Discuss contraception with individuals of child-bearing age (see page 37).
- Recommend vaccinations (see page 39).

* Many medications are either contraindicated or require dose adjustments as eGFR declines. We recommend checking individual product information, particularly before prescribing medications in CKD stage 4-5.

Lifestyle changes to help manage CKD

Lifestyle changes should always be considered as the first line management strategy for people diagnosed with CKD. Working with the individual to implement positive lifestyle changes in relation to: **S**moking, **N**utrition, **A**lcohol, **P**hysical activity (SNAP)[33], as well as adequate sleep, and stress management, can have a positive effect on CKD outcomes and delay the progression of disease.

The 5 As provide a key framework for addressing lifestyle changes.

Smoking / vaping

Target

- Don't smoke or vape. To aid smoking/vaping cessation, recommend counselling and if required, nicotine replacement therapy or other medication.

- Compared to people who have never smoked, the risk of developing CKD is increased by 27% for ever smokers, 34% for current smokers and 15% for former smokers.[34]
- Vaping has been associated with an increased occurrence of albuminuria and raised uACR levels.[35]
- Use of e-cigarettes has been linked to hyperuricaemia.[36]

Further information and resources

- Quitline - 13 7848.

Nutrition and diet

Target	
Dietary pattern	• Adopt a healthy dietary pattern that includes vegetables, fruit, wholegrains, nuts and legumes, dairy foods, lean meat, poultry, fish, and plant protein. • Healthy dietary patterns are associated with reduced risk of mortality and kidney failure,[37] developing CKD,[38] and progression of CKD[39]. • Consuming adequate fruits (2 serves /day) and vegetables (5 serves / day) can reduce the rate of kidney function decline, decrease body weight and blood pressure, and net acid production to manage metabolic acidosis in CKD.[40]
Fluid	• Make water your drink of choice. • There is no recommendation for the number of glasses of water that should be consumed daily for kidney health. It is recommended that people should drink water to satisfy thirst. • Sugar-sweetened beverages (SSBs) have shown to elevate the risk and progression of CKD and should be avoided.
Salt	• Salt intake should be reduced to <5 g per day. This may be achieved by adopting strategies, such as: – not adding salt in cooking or at the table. – choosing packaged foods with <120 mg sodium per 100 g. • People with a history of hyperkalaemia should not use salt substitutes and use caution with some reduced salt products, due to them containing high amounts of potassium chloride.
Ultra-processed foods and sugar	• All people with CKD should avoid ultra-processed foods high in fat, sugar, and salt, such as biscuits, cakes, packaged snack foods, takeaway foods, soft drinks, energy drinks, sports drinks, fruit juices and cordials.

- Individualised diets, tailored to cultural differences, food intolerances, cooking skills, food security, comorbidities (such as diabetes), and cost should be considered when recommending dietary options to patients with CKD and their families.
- For adults with diabetes and CKD, there is no one specific eating pattern that is superior. Individualised advice is recommended to help optimise glycaemic control.[41]
- Consider referral to an Accredited Practising Dietitian (APD), especially for help with implementing complex dietary changes, such as potassium restrictions, and if other chronic conditions such as diabetes, are present.

Further information and resources

- Kidney Health Australia Diet and Nutrition resources.
- Bush Tucker and Kidney Disease booklet.
- Easy Diet Diary Renal App: Available for download from your app store.
- Dietitians Australia: Find a dietitian
- Eat for health: Nutrient Reference values.

Alcohol

Target

- Reduce alcohol consumption – the less you drink, the lower your risk of harm from alcohol.[19]

- Australian guidelines recommend healthy men and women should drink no more than 10 standard drinks a week and no more than 4 standard drinks on any one day to reduce the risk of harm from alcohol-related disease or injury.[19]
- There are no specific recommendations about safe levels of alcohol consumption in people with CKD.

Further information and resources

- Australian guidelines to reduce health risks from drinking alcohol.

Physical activity

Target

- Be active on most days, preferably every day.
- Aim for 2.5 – 5 hours of moderate intensity activity across the week[17], or at a level that accounts for the individual's cardiovascular and physical health.[18]
- Some activity is better than none.
- Include muscle strengthening activities as part of daily activity at least twice per week.[17]

- Higher cardiorespiratory fitness levels, increased participation in physical activity and less time spent in sedentary pursuits are all associated with better outcomes in those living with CKD.[42]
- A person's overall health, heart health, comorbidities and physical capacity should be considered before increasing physical activity.
- Light to moderate intensity exercise is a safe starting point for most individuals. Vigorous intensity exercise should only be undertaken by those already doing this level of activity.[43, 44]

Further information and resources

- Physical activity and exercise guidelines for all Australians.

Weight management

Target

- Waist circumference <94 cm in men (<90 cm in Asian men) or <80 cm in women (including Asian women).
- BMI <25 kg/m^2 (<23 kg/m^2 Asian population[16]).

- Central obesity (measured by waist circumference) is a strong predictor of CKD progression.
- Obesity (BMI ≥30 kg/m^2) doubles the risk of developing CKD.[45]
- People with BMI ≥30 kg/m^2 may be more likely to develop albuminuria.[45]

Further information and resources

- Resources on Obesity and Overweight.

Pharmacological management of CKD

Clinical tip

Remember to code CKD correctly. Your practice software can help with medication considerations.

Medication considerations in CKD

- It is important to review medications that are excreted by the kidneys and avoid nephrotoxic medications in people with CKD.
- Dosage reduction or cessation of medications that are excreted by the kidneys is generally required once the GFR falls below 60 mL/min/1.73m^2.
- Home Medicines Reviews and Residential Medication Management Reviews support general practitioner (GP)/pharmacist collaboration and are funded by Medicare.
- Educate your patients to flag their CKD diagnosis with other providers and ensure that they are aware that having CKD can affect prescribing of medications.
- Ensure patients are aware of risk of AKI and have a sick day action plan. Refer to AKI sick day action plan on page 40 for further information.

Clinical tip

If patients become ill and are unable to maintain adequate fluid intake, they should be advised to withhold medications that increase the risk of kidney function decline and adverse events or have reduced clearance. Sulfonylureas, ACE inhibitors, diuretics, metformin, ARBs, NSAIDs, and SGLT2 inhibitors (SADMANS mnemonic) should be temporarily discontinued during acute illness, especially in the context of sepsis, hypovolaemia or hypotension, and recommenced when the condition stabilises.

Prescribe: medications that slow CKD progression and reduce cardiovascular risk

ACE inhibitor or ARB	Statin (+/- ezetimibe)	SGLT2 inhibitor*	Non-steroidal MRA*	GLP-1 RA*
• First-line treatment for all people with CKD. • Up-titrate to maximum tolerated dose to reduce albuminuria and kidney function decline.	Consider use in: • people with CKD (eGFR ≥15 mL/min/1.73m^2) and CVD risk ≥10% and • First Nations Australians with CKD and a CVD risk ≥5%.	• Recommended for use in people with CKD and proteinuria, with or without diabetes to reduce the risk of progressive decline in kidney function*. • Not recommended to initiate if eGFR <25 mL/min/1.73m^2.	• Indicated for use in people with CKD (with albuminuria) associated with type 2 diabetes. • Not recommended to initiate if eGFR <25 mL/min/1.73m^2 or in patients with a K+ >5.0 mmol/L.	• Indicated for use in people with CKD if they also have concomitant type 2 diabetes. • Not recommended for use in people with kidney failure.

* Check product information of relevant medication for eligibility criteria and recommended dosages.

Clinical tip

A reversible drop in eGFR is expected with the introduction of ACE inhibitors or ARBs. Check eGFR within 2 weeks following initiation and if reduction of eGFR is more than 25% below baseline value, cease the medication and consider referral to nephrologist.

Clinical tip

A reversible drop in eGFR is also expected with the introduction of SGLT2 inhibitors. The drop is at its greatest 4 weeks after initiation of therapy, after which the eGFR rebounds. Specific testing of eGFR for this purpose is not required. As SGLT2 inhibitors cause an osmotic diuresis, consider reducing diuretics and/or antihypertensive medications upon initiation of an SGLT2 inhibitor.

Reduce: medications excreted by the kidneys

Medications that may need to be started at a reduced in dose or ceased in patients with CKD include but are not limited to#

Anti-infective	Cardiovascular	Diabetes	Pain	Other
• famciclovir • nirmatrelvir • valaciclovir • certain antibiotics e.g., ciprofloxacin, trimethoprim, and sulfamethoxazole, aminoglycosides, nitrofurantoin	• apixaban • dabigatran • digoxin • rivaroxaban • sotalol • spironolactone	• acarbose • all gliptins except linogliptin • insulin • metformin* • sulfonylureas	• gabapentin • opioid analgesics • pregabalin	• allopurinol • benzodiazepines • colchicine • baclofen • duloxetine • escitalopram • solifenacin • fenofibrate • denosumab^ • lithium

Many drugs are contraindicated or not recommended as eGFR declines. Check individual product information for eligibility criteria and recommended dosage.

* Metformin should be used at a reduced dose if eGFR 30-60 mL/min/1.73m^2 and only under specialist supervision if GFR <30 mL/min/1.73m^2. It should be temporarily interrupted during periods of ill health and/or change in kidney function.

^ While dose reduction of denosumab is not required in CKD, the risk of hypocalcaemia increases with more advanced CKD. In patients with CKD stage 3-5, discuss with a nephrologist prior to initiation and monitor serum calcium closely.

Avoid: nephrotoxic medications

Commonly prescribed drugs that can adversely affect kidney function in CKD	Commonly prescribed drugs that should be avoided temporarily during a sick day (SADMANS)*
• lithium • aminoglycosides • NSAIDs/COX-2 inhibitors - beware of the 'triple whammy' (See clinical tip)	• **S**ulfonylureas • **A**CE inhibitors • **D**iuretics • **M**etformin • **A**RBs • **N**SAIDs • **S**GLT2 inhibitors

* As part of a sick day action plan, it is important that patients are advised to seek guidance from their healthcare professional on temporarily stopping medications during periods of illness.

Clinical tip

Patient safety: The **'triple whammy'**, a combination of **RAS blockade** (ACE inhibitor or ARB), **diuretic** and **NSAID or COX-2 inhibitor** (except low-dose aspirin) can result in acute kidney injury, especially if the patient is volume-depleted, or CKD is present. Ensure individuals on ACE inhibitor or ARB, plus diuretic blood pressure medication, are aware of the need to discuss appropriate pain relief medication with a GP or pharmacist.

Use of radiographic contrast agents in the context of CKD.[46]

Contrast-induced nephropathy (CIN) has long been observed in both experimental and clinical studies. However, recent observational studies have questioned the prevalence and severity of CIN following intravenous contrast exposure. The use of imaging requiring contrast needs to be considered in the context of relative benefit of the contrast-enhanced imaging test and risk of CIN.

We recommend the following CIN risk classification for **adults**:

- People with eGFR ≥45 mL/min/1.73 m^2 are at negligible risk for CIN.
- People with eGFR between 30 and 44 mL/min/1.73 m^2 are at an intermediate risk for CIN unless diabetes is present, which would further increase the risk.
- People with eGFR <30 mL/min/1.73 m^2 are at high risk for CIN.

Please note:

- eGFR may be overestimated in individuals with low muscle mass.
- Risk can be reduced by hydration prior to and after the imaging (including with-holding diuretics).
- Metformin should be withheld prior to contrast-enhanced imaging if possible.

Nephrotoxicity has been reported with gadolinium-enhanced MRI studies in people with kidney impairment.[47] This concern for toxicity was largely related to the association of nephrogenic systemic fibrosis (NSF) in patients with kidney impairment (eGFR <30 ml/min/1.73m^2). NSF is uncommon and rare with eGFR >30 ml/min/1.73m^2. The risk needs to be considered against the potential benefit of the imaging study.

Further information and resources

- Kidney Health Australia Health Professional Hub: How to guide – Sick Day Action Plan and Template.
- Australian Medicines Handbook (subscription required).
- NPS Medicinewise.
- Pharmaceutical Benefits Scheme (PBS).
- Therapeutic Goods Administration (TGA).

Care approaches

Whole-of-practice approach to CKD management

The management of CKD is a collaborative effort. A whole-of-practice approach involving the GP, primary healthcare nurse and practice staff, maximises the opportunity for best practice care to occur. Identification of a clinical lead, clinical governance, correct coding of CKD and implementation of e-health will all impact outcomes. As kidney function declines, and complications and comorbidities increase, a whole-of-practice approach facilitates optimal care.

Use of MBS Item Numbers

There are several MBS item numbers that can be claimed to assist with the detection and management of CKD. These include chronic disease management items, healthcare assessments, nurse reviews, and allied health visits. Visit mbsonline.gov.au for more details.

Culturally safe kidney care for First Nations Australians

Healthcare professionals should provide culturally safe care and understand the diverse factors that can impact kidney disease outcomes in First Nations Australians.

In line with the person's personal preference, include family and community in appointments, and liaise with Aboriginal and Torres Strait Islander Health Practitioners and interpreters[26].

Further information and resources

- CARI Guidelines: Recommendations for culturally safe kidney care in First Nations Australians.
- WA Centre for Rural Health - The University of Western Australia: Clinical Yarning eLearning Program
- Australian Indigenous HealthInfoNet.
- National Aboriginal Community Controlled Health Organisation (NACCHO).

Contraception and pregnancy in CKD[48, 49]

Parenthood planning is a core component of care for many individuals living with chronic kidney disease (CKD) and kidney failure. Pregnancy causes major impacts on the kidney and can be a "kidney stress test". Pregnancy may unmask undiagnosed CKD or worsen existing known CKD. Pregnancies with CKD are high risk, and the risk increases as CKD stage worsens. Even early-stage CKD confers a higher risk of important adverse outcomes, particularly hypertensive disorders of pregnancy. Fertility declines as CKD stage worsens, but ovulation may occur even with advanced kidney failure. Even being on dialysis is no guarantee an individual with CKD will not become pregnant. An unexpected pregnancy without preconception planning presents challenging choices to people with CKD and their partners.

Expert preconception planning is critical to optimise foetal outcomes and outcomes for the person giving birth. Informing individuals with CKD that pregnancy will be safer and more successful if planned with their clinical team may provide compelling incentive to use contraception and optimise health ahead of conception. Contraception should be considered for all sexually active people of childbearing age who have CKD. A particular focus should be on women and people with uteruses taking teratogenic medication, or those with uncontrolled hypertension or uncontrolled underlying kidney disease.

Responsibility for contraception implementation is likely to fall to the GP. Contraception choices will be determined by patient preferences, the clinical history, and comorbidities. In general, in higher risk people (on teratogenic medications, advanced CKD, uncontrolled primary disease, kidney transplant), highly effective contraception is recommended, rather than barrier contraception alone. Oestrogen-based contraception may not be ideal in people with hypertension, past thrombosis, proteinuria, or certain conditions (e.g., lupus). Intrauterine devices are generally safe in CKD.

Permanent strategies, i.e., partner vasectomy or tubal ligation should be considered for those who have completed their family or are not planning a pregnancy in the future.

Consultation with a high-risk pregnancy clinician (e.g., maternal medicine, obstetric physician, obstetric nephrologist) is recommended for people with CKD of any stage, who are planning a pregnancy or are pregnant. Ideally this should occur preconception, but definitely once pregnancy has been confirmed, to enable risk stratification and a pregnancy plan.

Considerations in older people

- Care of elderly people with CKD requires an individualised approach to address comorbidities, together with variability in functional status, life expectancy and health priorities.
- Relying on creatinine alone causes under-recognition of CKD.
- eGFR (which is adjusted for age) improves diagnostic accuracy.
- Dialysis therapy may not be associated with a survival advantage compared with non-dialysis comprehensive conservative care in elderly patients with two or more comorbidities.
- In older patients, treatment choice often has more effect on lifestyle than it does on mortality or morbidity.
- Utilise decision aid tools, such as Kidney Health Australia's the *'My Kidneys My Choice Decision Aid'*.

Clinical tip

An eGFR <60 mL/min/1.73m^2 is common in older people but is nevertheless predictive of significantly increased risk of adverse clinical outcomes and should not be considered physiological or age appropriate.

Appropriate referral

- Elderly patients with a stable eGFR ≥30mL/min/1.73m^2, microalbuminuria and controlled blood pressure can be managed successfully in primary care. The presence of anaemia may be related to CKD and a referral to a nephrologist may be indicated.
- Discuss management issues with a specialist by letter, email, or telephone in cases where it may not be necessary for the person with CKD to be seen by the specialist.
- The decline in eGFR can be variable and may depend on age, acute events, and other factors. Patients with eGFR <30 mL/min/1.73m^2 should be referred to a nephrologist and have a plan created for frequency of monitoring.

Medication considerations

- Diminished tolerance of side-effects and increased risk of adverse events is common with increased age.
- Reduced eGFR should lead to reduced doses of many drugs in the elderly.
- Polypharmacy is common in the elderly and increases the risk of falls, confusion, and functional decline.
- Home Medicines Reviews and Residential Medication Management Reviews support GP/pharmacist collaboration and are funded by Medicare item numbers.

Further information and resources

- Kidney Health Australia decision aid tools for people affected by CKD and health professionals: My Kidneys My Choice resources.

Vaccinations

These vaccinations are recommended for people aged 18 years and over with CKD:

- Influenza (yearly)
- Pneumococcal (5-yearly)
- COVID-19
- Herpes Zoster

Refer to the NIP for changes to recommendations on vaccinations that may be indicated or recommended for use in people with CKD.

Further information and resources

- The National Immunisation Program (NIP) Schedule.
- The Australian Immunisation Handbook.

Managing other kidney conditions

Acute Kidney Injury (AKI)[50, 51]

- AKI is common, especially in hospitalised patients, and is independently and strongly associated with increased morbidity and mortality.
- CKD increases the risk of AKI and in turn, an episode of AKI increases the likelihood of subsequent development of CKD, kidney failure and death, highlighting the need for ongoing surveillance.
- AKI is diagnosed either by detection of a sudden increase in serum creatinine, or with persistent oliguria (see below).
- Primary care professionals are in a unique position to identify people at increased risk of AKI and address potentially modifiable exposures to prevent the occurrence of AKI.

Risk factors for AKI[51]

Pre-existing risk factors	Potentially modifiable kidney insults

• CKD
• Other chronic diseases, e.g.:
  - diabetes
  - heart/lung/liver disease
  - cancer
  - anaemia
• Advanced age
	Pre-kidney:
	• Hypovolaemia
	• Blood loss
	• Hypotension
	• Shock
	Kidney:
	• Critical illness
	• Drug toxicity
	Post-kidney:
	• Obstruction

Identifying those at risk	How to diagnose AKI	What to do during an AKI episode	What to do after an AKI episode
• All people with CKD stage 3-5 are at increased risk of AKI. • Minimise use of NSAIDs and other potentially nephrotoxic drugs in people with CKD. • Early identification of people at risk with acute illness, and consider temporary cessation of ACE Inhibitor/ARB/ diuretics with hypovolaemia/ hypotension in line with sick day action plan.	• Increase in serum creatinine to ≥1.5 times baseline, which is known or presumed to have occurred within the 7 days prior, or • Significant reduction in urine output compared with normal output.	• Treat the cause. • Seek specialist advice early. • Systematic fluid assessment and medication review for all people at risk when acute illness occurs.	• Kidney Health Check after 3 months and then annually for subsequent 3 years. • Education and self-management to monitor and reduce risk of subsequent exposures. • Record in practice records as AKI (resolved).

Preventing AKI in individuals with CKD who are sick or dehydrated

If patients become ill and are unable to maintain adequate fluid intake (e.g., due to gastrointestinal upset or dehydration) they should be advised to withhold medications which will:

Increase risk of decline in kidney function	Increase risk for adverse events
• ACE inhibitors • ARBs • NSAIDs • Diuretics	• Metformin • Sulfonylureas • SGLT2 inhibitors

Mnemonic for drugs to be avoided on a sick day (SADMANS)

- **S** Sulfonylureas
- **A** ACE-inhibitors
- **D** Diuretics
- **M** Metformin
- **A** Angiotensin receptor blockers
- **N** Non-steroidal anti-inflammatory
- **S** SGLT2 inhibitors

Further information and resources

- Kidney Health Australia Health Professional Hub: How to guide – Sick Day Action Plan & Template.

Kidney cysts[52]

Simple cysts

Most simple kidney cysts are benign and do not require further investigation. They:

- are very common (not inherited) - prevalence ~10%
- are usually asymptomatic
- can occur with advancing age
- may be associated with background CKD
- do not cause kidney failure

Indications for further review and investigation:

- multiple cysts
- bilateral multiple cysts
- cysts with complex internal structure or solid components
- history of malignancy
- symptoms from cyst (discomfort, haematuria, infection)
- rapidly enlarging cysts

Polycystic kidney disease (PKD)

- Polycystic kidney disease (PKD) is a group of chronic kidney diseases with formation of multiple cysts in the kidney.
- The two main types of polycystic kidney disease are, Autosomal Dominant PKD (ADPKD) and Autosomal Recessive PKD (ARPKD), with ADPKD being much more common.
- PKD is the most common inherited kidney disease.
- Approximately 10% of people with ADPKD have no family history of the disease.[53]
- PKD is a common cause of CKD.

Consider a diagnosis of PKD if:

Age	Number of cysts shown on ultrasound
Aged 15-39 years	At least 3 in total
Aged 40-59 years	At least 2 in each kidney
Aged 60 years or older	At least 4 in each kidney

Treatments for ADPKD

Often the trajectory experienced by other family members provides some insight into their risk for progressive kidney disease. Early referral to Nephrologist is essential to ensure adequate treatment can be initiated. The medication 'tolvaptan' is listed on the PBS for the treatment of adults with early-stage CKD (stage 2 to 3) and rapidly progressing ADPKD. It has shown to slow the progression of cyst development and kidney disease in ADPKD. Refer to the PBS guidelines for guidance on which patients can be prescribed 'tolvaptan' and the relevant rules of prescribing.

Clinical management of autosomal dominant polycystic kidney disease (ADPKD)

1. Early referral to a nephrologist is recommended, so appropriate treatment can be initiated.
2. Assess risk for kidney failure based on family history and age at kidney failure.
3. Reduce kidney cyst growth and prevent eGFR decline and hypertension.
4. Evaluate for other kidney complications.
5. Consider genetic testing.
6. Magnetic resonant angiogram (MRA) to screen for intracranial aneurysms in high-risk individuals.

Usually, ADPKD is managed in consultation with the nephrology team.
See the CARI Guidelines website for specific guidelines for clinical management.

Further information and resources

- CARI Guidelines: Autosomal Dominant Polycystic Kidney Disease.
- Kidney Health Australia factsheet for people affected by CKD: Polycystic kidney disease.
- PKD Foundation of Australia.

Kidney stones[54]

- Kidney stones are one of the most common disorders of the urinary tract.
- The lifetime risk of developing kidney stones is 1 in 10 for Australian men and 1 in 35 for women. The risk increases with age and family history.[55]
- The most common types of stones are calcium oxalate and calcium phosphate.
- After having one kidney stone, the chance of a second stone is about 5-10% each year. About 30-50% of people with a first kidney stone will get a second one within five years, and then the risk declines.

Stone workup

- A general chemistry screen including serum uric acid, calcium, and parathyroid status.
- Stone analysis (when available).
- 24-hour urine volume and chemistries (including calcium, oxalate, citrate, and uric acid) are the mainstay of initial assessment and monitoring of response to interventions in adults.

Prevention of recurrence

- Typically, existing calcium stones cannot be dissolved.
- The goal of therapy is to reverse the abnormalities detected during the initial workup (e.g., low urine volume, hypercalciuria, hypocitraturia, and hyperoxaluria). Both dietary and fluid input changes and the use of medications may be necessary.
- Refer to an Accredited Practising Dietitian for a 3–6-month trial of diet and fluid changes before initiating drug therapy.
- Dietary changes to reduce calcium oxalate stones include:
  - increasing the fluid intake throughout the day (to maintain at least 2 L of urine per day).
  - increasing dietary potassium and phytate (e.g., nuts, beans) and maintain normal calcium intake.
  - decreasing the intake of oxalate, animal protein, sucrose, fructose, sodium, supplemental calcium.
- Drug therapy (depending on stone type) should be commenced if there is evidence of continued new stone formation, or if there is no or little improvement in the baseline urine chemistries with fluid and diet changes:
  - allopurinol to reduce hyperuricaemia.
  - citrate for hypocitraturia.

Acute management

- The acute management of a stone episode is usually undertaken in an emergency department with urologist involvement.
- The management of a stone episode, where the stone is known to be of a size able to be spontaneously passed (<5 mm), should include the use of an alpha blocker, such as prazosin or tamsulosin.

Clinical tip

Stone recurrence can be prevented in the majority of patients, who adopt a regimen that is devised after initial evaluation of the stone type and risk factors present in the individual.

Further information and resources

- Kidney Health Australia factsheet: Kidney Stones.

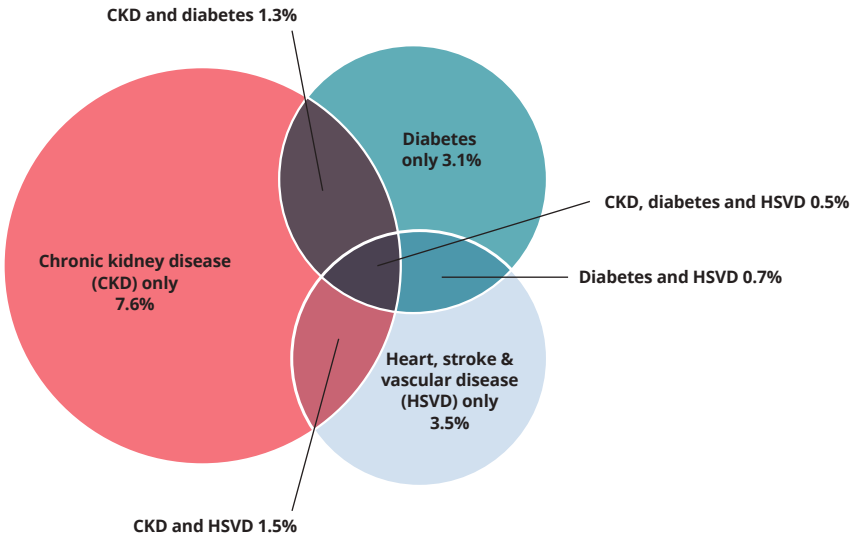
3

Managing CKD alongside other chronic conditions

Managing CKD in conjunction with other chronic conditions

CKD rarely occurs in isolation. In a primary care setting, it is very likely that individuals will have a CKD diagnosis that sits alongside one or more other chronic conditions. CKD shares many treatment goals and management strategies with other common chronic conditions, such as diabetes and cardiovascular disease. Taking a 'whole of person' approach and managing chronic conditions in conjunction with one another will lead to improved patient outcomes.

Prevalence of heart, stroke and vascular disease (HSVD), diabetes, CKD, and their comorbidity*[2]



*For persons aged 18 and over.

Adapted from: Australian Institute of Health and Welfare. (2023). *Chronic kidney disease: Australian facts.* Retrieved from https://www.aihw.gov.au/reports/chronic-kidney-disease/chronic-kidney-disease

CKD and cardiovascular disease

- All people with CKD have an increased risk of experiencing a cardiovascular event. This risk increases further and is high for people with moderate to severe CKD.[56]
- Both, a reduced eGFR and the presence of albuminuria are independent risk factors for cardiovascular disease (CVD).[29, 56]
- CKD is a more potent risk factor for CVD than diabetes.[57]
- Even early-stage CKD constitutes a significant risk factor for cardiovascular events and death, particularly in the presence of albuminuria.[58]
- For people with CKD, the risk of dying from cardiovascular events is up to 20 times greater than the risk of requiring dialysis or transplantation.[4]

Cardiovascular risk assessment in people with CKD

- The Australian CVD risk calculator can be used to estimate a person's cardiovascular risk.
- The CVD risk estimate represents the probability of having a cardiovascular event in the next 5 years.
- People with **moderate to severe CKD** (eGFR <45 mL/min/1.73m^2 or uACR >30 mg/mmol) are considered to have **pre-determined high risk** of experiencing a cardiovascular event in the next 5 years (≥10% probability).
- For people with eGFR 45-59 mL/min/1.73m^2 and/or uACR 3-30 mg/mmol, we recommend that their CVD risk is reclassified to a higher risk category to reflect albuminuria as a significant driver of CVD.
- Using the Aus cardiovascular risk calculator can be a helpful for determining meaningful and individualised levels of CVD risk.
- Clinical decisions based on cardiovascular risk can lead to improved health outcomes and be useful to educate and motivate patients.

Clinical tip

People with moderate to severe CKD (eGFR <45 mL/min/1.73m^2 or uACR >30 mg/mmol) are considered to have pre-determined high risk of experiencing a cardiovascular event in the next 5 years (≥10% probability). For people with eGFR 45-59 mL/min/1.73m^2 and/or uACR 3-30mg/mmol, we recommend that their CVD risk is reclassified to a higher risk category.

Further information and resources

- Kidney Health Australia Evidence Report: Make the Link: Kidney Diabetes and Heart.
- Kidney Health Australia Health Professional Hub: How to guide - Make the Link – CKD, Diabetes and CVD.
- CVD guidelines and Aus CVD risk calculator.
- Kidney Health Australia factsheet for people affected by CKD: Make the Link - CKD, Diabetes, Heart

CKD and diabetes

- One in three people with diabetes will develop CKD.[59]
- The presence of diabetes worsens the outcomes in all stages of CKD (cardiovascular outcomes, dialysis survival, and post-transplant survival).[60]
- Diabetes is a significant risk factor for CKD with 40% of cases of kidney failure caused by diabetes. This rises to over 70% for First Nations Australians.[15]
- Kidney disease is known to augment cardiovascular risk in diabetes.[61]

Diabetes treatment targets in people with CKD:	
BGL	• 6-8mmol/L fasting. • 8-10 mmol/L postprandial.
HbA1c	Generally: ≤53 mmol/mol (range 48-58); ≤7% (range 6.5-7.5)* • Target should be individualised according to patient circumstances (e.g., disease duration, life expectancy, important comorbidities, and established vascular complications). • HbA1c may not be a reliable indicator of glycaemic control in certain patient populations such as those with iron deficiency (elevated HbA1c) or anaemia (decreased HbA1c). * There may be situations where a lower target may be appropriate[62].

Management

- Provide advice on positive lifestyle changes (see page 29-32).
- Optimal blood glucose control significantly reduces the risk of developing microalbuminuria, macroalbuminuria and/or overt nephropathy in people with type 1 or type 2 diabetes.
- The definition of 'optimal' will vary depending on the balance between benefits and risks and the individual's priorities[62] (see Management of Type 2 Diabetes: A handbook for general practice, for individualised recommendations).
- Some medications may need to be reduced in dose or ceased in CKD (refer to tables on pages 34-35).
- When considering available diabetes treatment options, it is important to note that the presence of CKD may increase the risk of hypoglycaemia, particularly as CKD progresses.[63] The mechanisms relate to clearance of endogenous and exogenous insulin.
- Hypoglycaemia becomes more frequent as eGFR declines and medications may need to be adjusted accordingly.

Commonly used diabetes medications

Check individual product information and PBS listings for prescribing criteria and guidance. Indications and dosing guidance may have changed since publication.

Medication Class	CKD Dosing	Comments
Metformin	• For people with eGFR ≥60 mL/min/1.73m^2, use metformin at maximum dose required and monitor kidney function annually. • For people with eGFR <60 and ≥45 mL/min/1.73m^2, continue use and increase monitoring of kidney function (every 3-6 months). • For people with eGFR <30 mL/min/1.73m^2, only use under specialist supervision.	• Should be stopped temporarily during periods of illness due to potential risk of lactic acidosis.
SGLT2 inhibitors	SGLT2 inhibitors currently available in Australia: • Dapagliflozin – no dose adjustment required. – Initiation not recommended if eGFR <25 mL/min/1.73m^2. • Empagliflozin – no dose adjustment required. – contraindicated if eGFR <30 mL/min/1.73m^2 in people with diabetes. • Ertugliflozin – contraindicated in patients with CKD stage 4 or 5 or eGFR persistently <45 mL/min/1.73m^2. – kidney function should be monitored.	• Significant kidney and cardiovascular benefits conferred in people with CKD with and without diabetes.[64-69] • Possible side effects include, euglycaemic diabetic ketoacidosis (eDKA), thrush. • Cease when commencing kidney replacement therapy. • If prescribing for CKD or heart failure in absence of diabetes, dosing guidance may differ.
Non-steroidal MRA	• Finerenone – Initiation not recommended if eGFR <25 mL/min/1.73m^2 or serum K^+ > 5.0 mmol/L.	• Not currently indicated for diabetes management when CKD is not present. • Consider increasing Hyperkalaemia monitoring. • Stop, if serum potassium >5.5mmol/L. • Cease when commencing kidney replacement therapy.

Medication Class	CKD Dosing	Comments
Dipeptidyl DPP-4 inhibitors (gliptins)	• Linagliptin – no dose adjustment required for people with CKD. • Sitagliptin – reduce dose if eGFR <45 mL min/1.73m^2. • Saxagliptin – reduce dose if eGFR <45 mL/min/1.73m^2 – use with caution if eGFR 15-30 mL/min/1.73m^2. – not recommended if eGFR <15 mL/min/1.73m^2 or requiring dialysis. • Vildagliptin – no dosage adjustment required if eGFR >60 mL/min/1.73m^2. – reduce dose if eGFR 15-59 mL/min/1.73m^2. – limited experience in individuals with kidney failure, thus should be used with caution in this group. • Alogliptin – Reduce dose if eGFR <50 mL/min/1.73m^2.	• Not suitable for people with history of pancreatitis. • Risk of hypoglycaemia can be increased if prescribed with sulphonylureas.
Sulfonylurea (SU)	• Sulfonylureas – reduce dose if eGFR <30 mL/min/1.73m^2. • Glibenclamide – contraindicated if severe reduction in kidney function.	• As eGFR declines, risk of hypoglycaemia increases.
GLP-1 receptor agonist	• Semaglutide – no dose adjustment required in CKD. – not recommended for use in patients with kidney failure (eGFR <15 mL/min/1.73m^2). • Dulaglutide – no dose adjustment required if eGFR* >15 mL/min/1.73m^2. – not recommended for use when eGFR* <15 mLmin/1.73m^2.	• Potential cardiovascular and kidney benefits.[70] • Limited data on use in people with eGFR <30 mL/min/1.73m^2).
Insulin	Dose adjusted to blood sugar level	• As eGFR declines, risk of hypoglycaemia increases.

* For consistency, we interchanged Creatinine clearance (CrCl) with eGFR. Please note that CrCl overestimates glomerular filtration rate (GFR) and estimated CrCl differs from estimated GFR. The Cockcroft-Gault equation estimates CrCl in mL/min while the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation estimates GFR in mL/min/1.73m^2.[71]

Further information and resources

- RACGP Management of type 2 diabetes: A handbook for general practice.
- Kidney Health Australia factsheet for people affected by CKD: Diabetic kidney disease.
- KDIGO guidelines: Diabetes in CKD.
- Australian Diabetes Society.

CKD and hypertension

Hypertension is both a cause and a complication of CKD and can be difficult to control. The risks of uncontrolled hypertension include, progression of kidney disease and increased risk of coronary heart disease and stroke. Hypertension should be considered as part of cardiovascular risk (see page 48).

Hypertension treatment targets

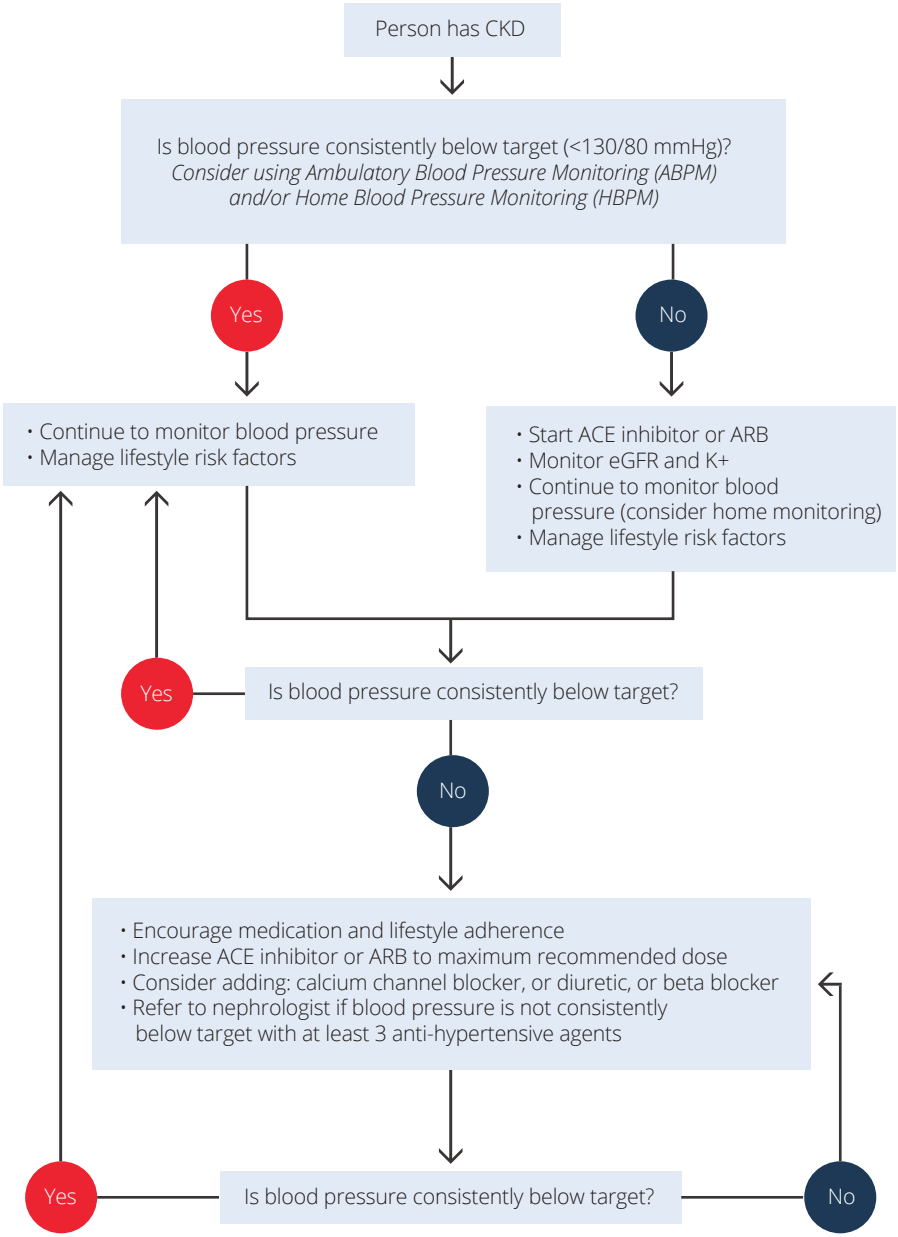
Who?	Target
All people with CKD	**Maintain blood pressure consistently below 130/80 mmHg***

* Some evidence and clinical guidelines suggest aiming for a lower blood pressure target (systolic BP <120mmHg) in people with CKD who are at high CVD risk may improve outcomes.[72-75]

Aiming for a systolic blood pressure of <120 mmHg may be appropriate in certain individuals who are at very high cardiovascular risk.

Lower blood pressure targets need to be balanced with an increased risk of side effects including: increased risk of falls due to hypotension, electrolyte abnormalities and episodes of AKI.

Algorithm for management of hypertension in people with CKD



Management

- Reducing blood pressure to below target levels is one of the most important goals in the management of CKD.
- Lifestyle changes should always be advocated and can have a significant effect on blood pressure (see pages 29-32) for guidance on basic lifestyle advice).
- Target BP <130/80 mmHg ideally with an ACE inhibitor or ARB alone or in combination with CCB, diuretic, or beta blocker.
- When treatment with an ACE inhibitor or ARB is initiated, the GFR can decrease, and potassium levels can rise (see pages 34 & 68 for more information).
- If the serum potassium concentration is greater than 6 mmol/L despite dose reduction, diuretic therapy, and dietary potassium education, then ACE inhibitor, ARB, and steroidal and non-steroidal MRAs should be stopped.
- Multiple medications (often 3 or more drugs) are needed to control hypertension adequately in most people with CKD.
- Consider sleep apnoea as a cause of resistant hypertension.

Blood Pressure Monitoring

- 24-hour ambulatory blood pressure monitoring (ABPM) or home blood pressure monitoring (HBPM) have been shown to better correlate with target organ damage and cardiovascular mortality and morbidity when compared to office BP measurements.
- Use of ABPM or HBPM can also aid the diagnosis of masked hypertension, over treatment (hypotension) and monitor response to antihypertensive treatment. The use of ABPM is rebated by MBS, if done to confirm the diagnosis and before starting medication.
- HBPM when combined with education may increase adherence and improve overall blood pressure control.
- Where feasible HBPM should be considered to aid in the diagnosis and management of hypertension.
- Sphygmomanometers requiring calibration must be serviced regularly according to the manufacturer's instructions.

Commonly used anti-hypertensive medications

Medication	Notes on use in people with CKD
Renin-Angiotensin System (RAS) inhibitors - e.g., ACE inhibitors or ARBs	**Essential part of the best care approach for many patients in all stages of CKD.** • Based on many clinical trials demonstrating their effectiveness in decreasing proteinuria and delaying CKD progression, the use of RAS inhibitors is recommended as the first-line pharmacologic strategy for patients with CKD and proteinuria with and without diabetes. • They cause a reduction in glomerular blood flow, and GFR can decline when treatment is initiated. • Provided the reduction is less than 25% within two weeks of starting therapy, the ACE inhibitor or ARB should be continued. • If the reduction in GFR is greater than 25% below the baseline value, the ACE inhibitor or ARB should be ceased, and a referral to a nephrologist considered. • In general, combined therapy with ACE inhibitor and ARB is not recommended. • Caution should be exercised if baseline potassium is ≥5.5 mmol/L, as rises in serum potassium of approximately 0.5 mmol/L are expected (see page 68). • ACE inhibitors or ARBs can be safely prescribed at all stages of CKD and should not be ceased as GFR progressively declines but continued if tolerated.[76]
Calcium channel blockers	May be used for people with angina, the elderly and those with systolic hypertension.
Diuretics (e.g., thiazides and loop diuretics)	• Loop diuretics are effective in all stages of CKD including when GFR is severely reduced to <30 mL/min/1.73m^2. – Typical doses are 20-120 mg/day, but higher doses (up to 500 mg/day) may be required, especially at lower levels of eGFR. – When more than 80 mg/d is required, the efficacy is improved by dividing the daily dose. • Thiazides can be effective at low levels of eGFR, particularly in combination with loop diuretics. For more information on managing oedema, see page 67.

Medication	Notes on use in people with CKD
Beta-blockers	Beta-blockers are useful agents for blood pressure control in people with CKD. For use of beta-blockers in heart failure, refer to the National Heart Foundation Australia guidelines.[77]
Steroidal MRA	Steroidal MRAs may be used for people with concomitant heart failure or hypertension. Use with caution in CKD and in combination with RAS blockade, due to risk of decline in eGFR and hyperkalaemia. Non-steroidal MRAs can be safely used in the setting of type 2 diabetes and CKD. Steroidal and non-steroidal MRAs should not be prescribed together. For both agents, it is recommended to monitor potassium carefully.

Clinical tip

ACE inhibitors, ARBs, SGLT2 inhibitors and diuretics should be temporarily discontinued during acute illness, especially in the context of sepsis, hypovolaemia, or hypotension, and recommenced when the condition stabilises.

Clinical tip

ACE inhibitors and ARBs cause a reversible reduction in glomerular blood flow and GFR can decline when treatment is initiated. Check eGFR within 2 weeks following initiation. Provided the reduction is less than 25% within two weeks of starting therapy, the ACE inhibitor or ARB should be continued. If the reduction in GFR is greater than 25% below the baseline value, the ACE inhibitor or ARB should be ceased, and referral to a nephrologist considered.

Further information and resources

- St Georges Community and Health Services: Home Blood Pressure Monitoring.
- KDIGO guidelines: Blood Pressure in CKD.
- Kidney Health Australia factsheet for people affected by CKD: Blood pressure and CKD.

CKD and heart failure

CKD commonly occurs with heart failure (HF), which is a common cause for hospitalisation, morbidity, and mortality in people with kidney disease.[78]

Cardiorenal syndrome describes a number of conditions where an acute or chronic dysfunction in the heart or kidneys leads to the dysfunction of the other organ.[79]

Management:

- Input from an interdisciplinary team, including a HF and kidney specialist may be necessary when providing care for people with concominant HF and CKD.[78]
- Medical management of CKD in the context of HF can be challenging due to the need to balance the differing effects of medications on both the kidney and the heart.[79]
- Medications may need to be increased or decreased, or temporarily stopped, especially when patients are unwell.
- Remembering the SADMANS mnemonic is of particular importance, as well as the restarting of all appropriate and tolerated medication, once the person recovers from any acute episodes of decline. (See pages 41-42 for further information on sick day management).
- It is important to consider patient wellbeing when treating a person with concomitant HF and CKD. A higher serum creatinine as a results of fluid management in HF may be deemed acceptable if it aids the patient's ability to breathe normally.
- Use of cardio-selective beta-blockers, renin-angiotensin system inhibitors (RAS inhibitors), angiotensin receptor-neprilysin inhibitors (ARNIs)[80] and MRAs in patients with HF and CKD stages 1–3 have shown both symptom and outcome benefits.[78] Nevertheless, due to the increased risk of hyperkalaemia and concerns regarding kidney function decline, ARNIs, RAS inhibitors and MRAs are often sub-optimally prescribed.[78]

Medication	
RAS inhibitors **ARNIs** **MRAs**	Should be used with usual advised precautions, i.e., associate decline in eGFR >25% with appropriate dose adjustments and/or stopping medication(s) temporarily and restarting at low doses, up-titrating slowly to tolerated dose. • ARNIs should not be prescribed together with an ACE inhibitor. • Steroidal MRAs should not be used together with non-steroidal MRAs. Monitor potassium carefully.
SGLT2 inhibitors	Use of SGLT2 inhibitors is recommended in the management of HF in patients with CKD of any stage, where indications for use of SGLT2 inhibitors are met. • Initiating an SGLT2 inhibitor in patients with an eGFR <25 mL/min/1.73m^2 is not recommended due to limited evidence.
IV iron	• Use of IV iron therapy is recommended in the management of HF in patients with CKD of any stage, where indications for use of IV iron are met.

4

Common issues in CKD

Early detection and intervention have been shown to reduce the progression of CKD and its complications. It is essential to regularly check for the known complications of CKD and to monitor treatment targets.

Acidosis

People with eGFR <30 mL/min/1.73m^2 are at increased risk of metabolic acidosis. The main factor is decreased kidney acid excretion compounded by a reduction in bicarbonate production. Acidosis contributes to demineralisation of bone and increased protein degradation, which may be associated with increased morbidity.

Management

- Supplementation with sodium bicarbonate (840 mg capsule) may be considered in people with acidosis:
  - typical starting dose would be 1 capsule od or bd, increasing up to 2 tablets bd if needed, and titrating to normalise the HCO3 level.
  - higher doses can be prescribed but carry a higher risk of fluid overload.
- Increased sodium load may worsen blood pressure control.
- A base-producing diet of predominantly fruits and vegetables can improve metabolic acidosis and slow down kidney function decline.[81, 82]

Albuminuria[32]

Albuminuria is an important prognostic feature in CKD. The degree of albuminuria relates to the severity of the kidney disease and likelihood of progression to kidney failure. The amount of albuminuria can be reduced significantly with an ACE inhibitor or ARB agent, SGLT2 inhibitor and non-steroidal MRA (the latter in the context of type 2 diabetes). Reduction in the amount of albuminuria is associated with improved outcomes.[83]

Target:

uACR reduction of at least 30%.[20-23]

Management

Stepwise approach for treating persistent macroalbuminuria:

1. Start an ACE inhibitor or ARB if not already using for hypertension.
2. Up-titrate ACE inhibitor or ARB to maximum tolerated dose (monitoring hypotension or hyperkalaemia).
3. Add an SGLT2 inhibitor where indicated.
4. If the person has type 2 diabetes, consider adding a non-steroidal MRA to reduce CKD progression and CVD events.
5. If the person has type 2 diabetes, consider adding a GLP-1 RA. Early evidence suggests that GLP-1 RA may have benefits on kidney and cardiovascular outcomes in people with type 2 diabetes.[70, 84]

Anaemia[85]

- Anaemia in CKD is related to:
  - reduced erythropoietin production by the kidney;
  - resistance to the action of erythropoiesis stimulating agents (ESA);
  - reduced absorption of iron.
- Anaemia related to CKD usually starts to develop when the GFR is less than 60 mL/min/1.73m^2. The prevalence of anaemia increases with decreasing GFR.

Target:

Hb 100 – 115 g/L.

Prior to commencement of ESA: Trial iron supplementation, maintaining ferritin >100 mcg/L; TSAT >20%

Once ESA commenced: Maintain ferritin 200-400 mcg/L; TSAT >20%

Management

- Iron studies should be conducted in people with stage 3-5 CKD as part of their regular assessment under the orange and red clinical action plans (see pages 27-28). This allows for earlier identification and treatment of anaemia.
- In people with CKD, other forms of anaemia should be considered and excluded:
  - Iron deficiency (absolute and functional) is a common cause of anaemia in people with CKD.
  - If absolute iron deficiency is identified, causes including GI blood loss should be considered and excluded.
  - Vitamin B12 and folate levels should be checked and corrected if deficient. Note, long-term use of proton pump inhibitors (PPI) is associated with vitamin B12 deficiency.
- Thyroid stimulating hormone should be assessed and hypothyroidism treated if present.
- Both significant hyperparathyroidism and systemic inflammation may contribute to anaemia and may cause refractoriness to erythropoietin therapy.
- Ensure adequately iron replete with either oral or IV iron.
- Treatment with ESA must be managed by a nephrologist. There are several ESAs currently available for this indication in Australia. All are available as pre-filled syringes and are usually administered subcutaneously to those with CKD or on peritoneal dialysis patients and intravenously to those on haemodialysis.
- For further guidance on anaemia in CKD refer to the CARI guidelines at www.cari.org.au

Cognitive decline

- It is important to assess cognition in people with CKD.
- Cognitive impairment is common in people with CKD and prevalence increases with CKD severity.
- Cognitive impairment is an important factor when approaching kidney failure as it will influence treatment choices and decision-making about future care.

The presence of CKD:

- Can affect global cognition, attention, memory and executive functions.
- Independently contributes to a decline in physical and cognitive functions in older adults.
- Can double the risk for physical impairment, cognitive dysfunction, and frailty in those >70 years.
- Is a risk factor for 'accelerated aging'.

Management

- Cognition affects many aspects of CKD care.
- Things to consider in primary care assessments are:
  - Screen for cognitive impairment in CKD, e.g., with Mini-Mental State Examination (MMSE) or Montreal Cognitive Assessment (MoCA)
  - Safety
  - Medication adherence
  - Medication review
  - Falls
  - Risk of delirium
  - Association with depression
  - Self-care issues and engagement with care
  - Frailty

Depression[86]

Depression can affect 1 in 5 people with CKD, and 1 in 3 individuals on dialysis. Depression in people with CKD has detrimental effects on mortality, rates of hospitalisation, medication and treatment adherence, nutrition, and overall Quality of Life. Treatment of depressive symptoms in people with CKD has the potential to improve health outcomes.

Management

- Screen regularly and maintain a high level of clinical awareness for depression. Consider use of DASS-21 or Kessler K10.
- Modifiable causes of depression that are commonly experienced by people with CKD (e.g. insomnia, medication side-effects, inadequate dialysis) should be considered and excluded.
- Treatment of persistent depressive symptoms involves a combination of non-medication therapies (e.g. education, cognitive behavioural therapy, exercise programs) and antidepressant medication.
- Selective serotonin reuptake inhibitors (SSRIs) have established safety in people with CKD.[86] (For a detailed list of the most common classes of antidepressant medications with suggested dosing in kidney impairment, and potential adverse effects refer to the article referenced).
- In patients with chronic conditions, depression and anxiety is often an impairment to their self-management strategies. Consider a General Practice Mental Health Care Plan referral to a psychologist for psychological support.

Further information and resources

- Head to Health - information on mental health for individuals and health professionals.
- General Practice Mental Health Standards Collaboration - resources for health professionals.
- Beyond blue - information for individuals.
- Black Dog Institute - information and resources for individuals and health professionals.
- Kidney Health Australia factsheet for people affected by CKD: Depression and Chronic Kidney Disease.

Haematuria

- The most common causes of haematuria are non-glomerular conditions such as menstrual contamination or urological conditions (urinary tract infection (UTI), kidney calculi, prostatic disease, or urinary tumours).
- Visible (or macroscopic) haematuria must always be investigated.
- Haematuria due to intrinsic kidney disease is called glomerular haematuria.
- Persistent haematuria, or haematuria found in conjunction with other indicators of kidney damage necessitates investigation.
- Under the age of 40, isolated haematuria (haematuria without albuminuria, reduced GFR, or urinary tract malignancy) is usually due to an underlying glomerulonephritis with a low propensity for progression. Annual follow-up is recommended to monitor for progressive disease.[87]

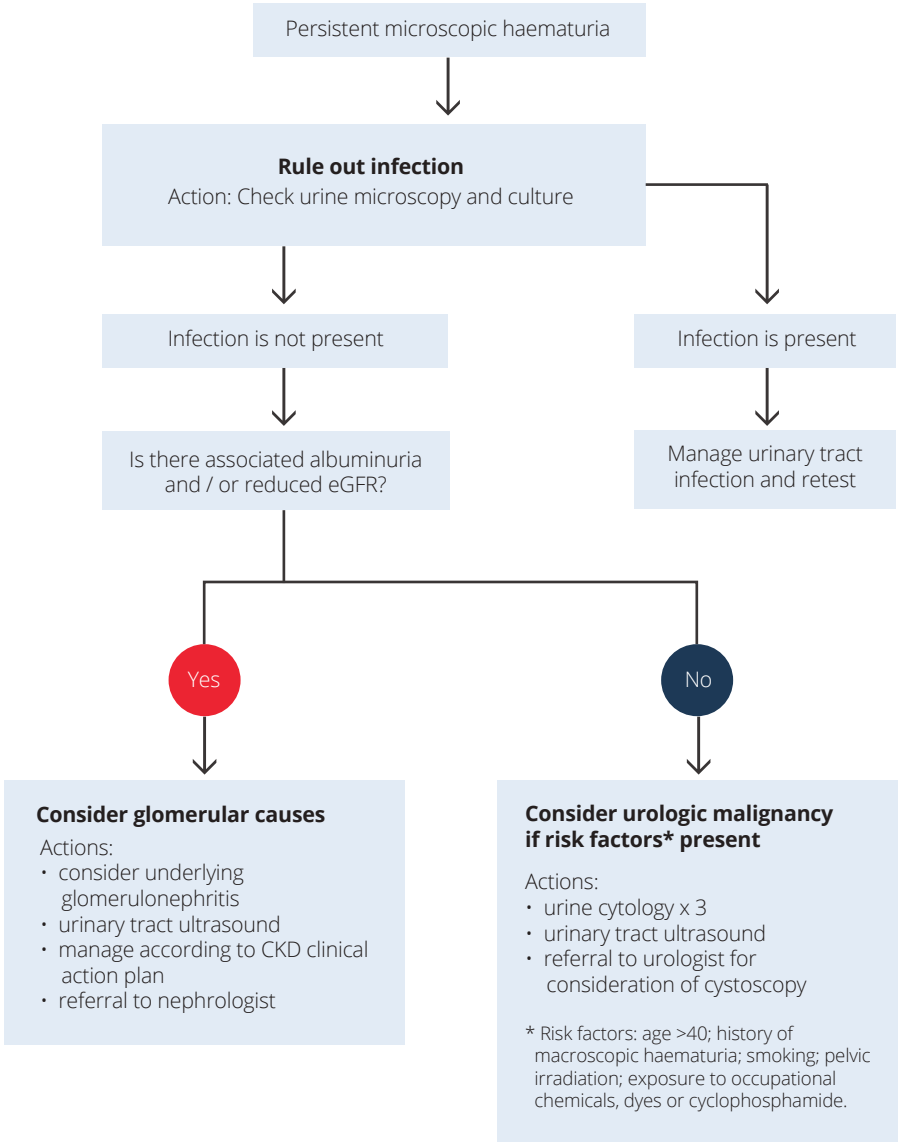
Assessment[88]

- If a dipstick is positive for haematuria, results should be confirmed by performing a urine analysis with microscopic evaluation of the urinary sediment of a freshly voided, clean-catch, midstream urine sample.
- Urine microscopy can sometimes be used to determine the origin of the blood – glomerular versus non-glomerular.
- Non-glomerular bleeding warrants urological investigation.
- Glomerular bleeding should prompt a Kidney Health Check.

Management

- Persistent microscopic haematuria, with or without albuminuria, should prompt investigation for urinary tract malignancy in appropriate age groups.[87]
- Persistent haematuria in the absence of albuminuria should be followed up annually with repeat testing for haematuria, albuminuria, eGFR and blood pressure monitoring as long as the haematuria persists.[87]
- Family members should also be screened for haematuria.

Algorithm for the management of persistent microscopic haematuria



Hyperuricaemia

- Hyperuricaemia is common in patients with metabolic syndrome and metabolic syndrome is common in people with CKD.
- Certain dietary factors can stimulate uric acid production, including purine-rich meats (especially seafood, organ meats), beer (because of the alcohol and yeast content), and fructose (including fruits, honey, sugar, and high fructose corn syrup).
- Other stimuli for uric acid production include ischaemia, heat stress, and conditions associated with rapid cell turnover (e.g., tumour lysis syndrome).
- Uric acid is also generated intracellularly under conditions in which aldose reductase is induced, such as with high glycaemic carbohydrates, salty foods, and dehydration.[89]
- Hyperuricaemia is associated with hypertension, gout, non-alcoholic fatty liver disease, CKD, and cardiovascular diseases due to increased oxidative stress, inflammation, and apoptosis.

Management

- In the presence of gout, uric acid lowering therapy may be indicated. Since allopurinol and its metabolites are excreted by the kidney, impaired kidney function may lead to retention of the drug and/or its metabolites with consequent prolongation of plasma half-lives. When initiating allopurinol in people with CKD, a starting dose of 50-100 mg/day is recommended depending on the stage of CKD. [90]
- Colchicine may be used to manage an acute flare of gout, however a reduction in the size of individual doses, an increase in the interval between doses or a reduction in the total daily dosage may be necessary in patients with CKD. Specifically, it is recommended that dosage be reduced by half if eGFR <50 mL/min/1.73m^2 and that colchicine not be used if the patient's eGFR <10 mL/min/1.73m^2.[91] A short course of prednisolone can be helpful in this instance. Low dose urate-lowering therapy (allopurinol 50 mg/d) may be commenced during the acute attack, in addition to adequate treatment for the acute attack.
- Dietary approaches that lower insulin resistance also reduce serum urate.[92, 93, 94]
- In the absence of acute gout, pharmacological lowering of uric acid with allopurinol is not recommended.[95]
- Treatment with allopurinol does not slow the decline in eGFR in patients with CKD alone nor CKD with diabetes[96].

Further information and resources

- CARI Guidelines: Urate-lowering therapy for people with chronic kidney disease.

Hyperkalaemia

Target:
Potassium ≤6.0 mmol/L.

In CKD, excretion of potassium (K+) in the urine is impaired. Potassium levels may also rise with the use of ACE inhibitors or ARBs used to treat hypertension or with use of steroidal or non-steroidal MRA. Potassium levels consistently above 6.0 mmol/L are of concern and should be managed. Hyperkalaemia, especially potassium levels >6.5 mmol/L, predisposes to cardiac arrhythmias.

Management

Potassium 6.0 – 6.5 mmol/L:

- Avoid ultra-processed foods.
- Low K^+ diet (discuss with an Accredited Practising Dietitian).
- Correct metabolic acidosis (Normalise serum HCO_3 via dietary and/or pharmacological intervention).
- Consider commencing potassium wasting diuretics (e.g., thiazides).
- Avoid salt substitutes which may be high in K^+.[97]
- Consider a cation exchange resin (e.g., sodium polystyrene sulfonate) or a non-absorbent cation exchange polymer (e.g., patiromer) for eligible patients (refer to PBS criteria).
- Cease ACE inhibitor, ARB, steroidal MRA, non-steroidal MRA if K^+ persistently >6.0 mmol/L and not responsive to above therapies.[87]

Potassium >6.5 mmol/L:

- Refer to nearest Emergency Department if K^+ >6.5 mmol/L due to the risk of arrhythmia.

Lipids

People with CKD have a characteristic lipid pattern of hypertriglyceridaemia and low HDL cholesterol levels but normal LDL cholesterol levels.[98] Dyslipidaemia is more severe in individuals with albuminuria, particularly those with nephrotic syndrome.

- No target lipid level is recommended.

Management[99]

- In adults with newly identified CKD, evaluation with a fasting lipid profile is recommended.
- Consider secondary causes and specialist evaluation if severely elevated fasting lipid levels (LDL-cholesterol >4.9 mmol/L or triglycerides >11.3 mmol/L).
- Follow-up measurement of lipid levels is not required for the majority of patients.
- Statin (+/-ezetimibe) for people with CKD (eGFR ≥15 mL/min/1.73m^2) and CVD risk ≥10%, and for First Nations Australians with CKD and CVD risk ≥5%.
- Lifestyle advice if hypertriglyceridaemia is present.

Further information and resources

- CARI Guidelines: Management of cholesterol-lowering therapy in people with chronic kidney disease.

Malnutrition[40]

Poor food intake due to anorexia in CKD can lead to malnutrition. See page 30 for more information on nutrition and CKD.

Management

- Dietary advice (refer to an Accredited Practising Dietitian).
- Malnutrition screening tools may be useful to identify those with unintentional weight loss and poor appetite.

Further information and resources

- MUST Malnutrition Universal Screening Tool (for adults).[100]
- Malnutrition Screening Tool (MST).[101]
- MNA-SF Mini- Nutritional Assessment short form (for people aged 65 and over).[102]

Mineral and bone disorder[32, 103, 104]

- Changes in the metabolism of calcium, phosphate, parathyroid hormone and vitamin D typically start to occur once GFR ≤60 mL/min/1.73m^2.
- As kidney function decreases, the clearance of phosphate by the kidneys is diminished, leading to higher serum phosphate levels.
- Levels of calcitriol, the most active form of vitamin D, fall because kidney function is required for its synthesis. Calcium levels may fall as a result of less vitamin D dependent calcium uptake from the gastrointestinal tract.
- The combined effects of higher phosphate, lower calcium and lower vitamin D levels all serve to stimulate parathyroid hormone (PTH) production. Elevated PTH levels increase the reabsorption and release of mineral from bone.
- These changes are associated with an increased risk of fracture and cardiovascular mortality, perhaps mediated by accelerated vascular calcification.

Management

- The management of CKD Bone Mineral Disorder (CKD-BMD) is complex and usually occurs via a nephrologist.
- CKD-BMD does not usually need to be tested for or addressed in CKD stage 1-3. In patients with CKD stage 3-5 with evidence of CKD-MBD and/or risk factors for osteoporosis, we suggest BMD testing to assess fracture risk if results will impact treatment decisions. Lower Dexa BMD predicts incident fractures in patients with CKD stage 3-5. No specific anti-resorptive is preferred.
- In patients with CKD stage 3-5, treatments of CKD-MBD, such as denosumab, should be discussed with a nephrologist prior to initiation. Serial serum calcium, phosphate and PTH are helpful in determining treatment.
- The risk of hypocalcaemia following denosumab increases with more advanced CKD and serum calcium should be closely monitored.
- Aim for a serum phosphate towards normal ranges (consider referral to a kidney dietitian to reduce phosphate in diet) and avoid hypercalcemia. The optimal level for PTH in the setting of CKD is not known.
- In early CKD, vitamin D level may be monitored and replaced if deficient. With more advanced CKD, calcitriol is preferred in the presence of hypocalcaemia due to the inability of the kidney to hydroxylate 25 vitamin D.
- As CKD is associated with vascular calcification, care is required not to overload with calcium supplementation.[105]

Muscle cramps

Muscle cramps are unpleasant and often reported in more advanced CKD. They may be related to electrolyte disorders, hypocalcaemia, and volume depletion.

Management

- Treatment is directed towards correcting electrolyte abnormalities.
- There is no evidence to show that magnesium supplementation effectively reduces muscle cramps.

Oedema

Fluid retention and overload may become a problem with worsening CKD severity. Oedema is rarely caused by early-stage CKD alone (except in nephrotic syndrome) and is more a feature of advanced stage CKD.

- Oedema may manifest most commonly as ankle (pedal) oedema.
- The clinical assessment should include blood pressure, respiratory examination when assessing patients with ankle oedema.
- Hypertension is common in fluid overload.
- Pulmonary oedema may be a feature of more advanced CKD.
- Ascites may be seen in severe fluid overload.
- Biomarkers such as brain natriuretic peptide (BNP) to assess heart failure may be unreliable in patients with CKD. Note, BNPs are not covered by the MBS.

The potential causes of oedema in patients with CKD to consider are:

- CKD – reduced water excretion and reduced urine output
- Nephrotic syndrome (urine protein loss and low blood albumin)
- Medications (calcium channel blockers nifedipine, steroids)
- Sodium retention and/or excess sodium intake
- Congestive heart failure
- Liver disease and low albumin
- Lymphoedema
- Vascular causes including deep vein thrombosis (DVT)
- Dependent oedema (gravity, poor mobility)

Ankle oedema

- Mild ankle oedema that is not symptomatic may be managed conservatively with raising legs, using stockings and moderate sodium restriction.
- Calcium channel blockers are a common cause of ankle oedema, which may warrant consideration of an alternative antihypertensive agent.
- Diuretic therapy with loop and thiazide diuretics should be used for treating ankle oedema only after assessment of volume status has occurred.

Diuretic resistance may occur in later stages of CKD – diuretic doses may need to increase.

Refractory oedema in advanced CKD is usually an indication to commence dialysis.

Pain management

Pain is a common symptom in patients with advanced CKD and kidney failure, and it can impact significantly on Quality of Life and function.

The approach to management includes a detailed history and examination to determine the specific pain syndrome e.g., diabetic peripheral neuropathy, or osteoarthritis.

Localised pain

Localised pain (nociceptive, soft tissue) may be treated with topical therapies like herbal liniment, or a topical massage cream such as capsaicin or salicylates, or a gel based NSAID.

Severe pain

Severe pain requires systemic treatment. Following the WHO analgesic ladder is recommended:

- **Step 1: Non-opioid analgesia**
  - Regular paracetamol is safe in kidney failure. Use 1g qid if needed, as the first line and background treatment.
  - Systemic NSAIDs are best avoided.
- **Step 2: Weak opioids**
  - Codeine and tramadol can be used cautiously in people with CKD.
  - For people with eGFR between 20-50 mL/min/1.73m^2, dosing is as the same as for people with normal kidney function, but with close monitoring of kidney function.
  - Codeine and tramadol <u>are not</u> routinely used in people with advanced CKD and must be avoided when eGFR <20 mL/min/1.73m^2, as accumulation of the drugs are common with serious side effects.

- **Step 3 Strong opioids**
  - Many short-acting and long-acting strong opioids can be used in people with CKD with appropriate dose adjustment. Treatment should be individualised according to the person's medical history and commenced in consultation with relevant specialist.

Note regarding use of Morphine:

- For people with eGFR 20-50 mL/min/min/1.73m^2, use 75% of normal dose.
- Should be avoided in kidney failure, especially advanced, i.e., if eGFR <20 mL/ min/min/1.73m^2, as both dose and intervals require adjustment, making it challenging to use.

Neuropathic/nerve pain

The following medications can be used as first-line treatments, or as an adjunct in nociceptive pain management, according to the WHO analgesic ladder.

1. Gabapentinoids (gabapentin or pregabalin)
   - Used in restless legs syndrome and uraemic pruritus.
   - Dose adjustment is recommended in patients with reduced kidney function and/ or those undergoing haemodialysis. Refer to product information for specifics.
   - Main side effects include drowsiness, ataxia, clumsiness, blurred vision.
2. Tricyclic antidepressants
   - Amitriptyline 10 mg nocte. Titrate up to 25 mg nocte after 3-7 days, and to 50 mg nocte after 1-2 weeks.
   - If no efficacy at 50 mg nocte, consider other alternatives.
   - Common side effects include drowsiness, dry mouth, and constipation.
3. Duloxetine (SNRI)
   - Duloxetine has evidence for efficacy in painful diabetic peripheral neuropathy.
   - Use 30 mg daily.
4. Other medications
   - Other medications useful for neuropathic pain include, lignocaine, mexiletine, and methadone, with pain team or palliative care guidance.

Further information and resources

- St George Hospital Community and Health Service: Pain.

Pruritus[106, 107]

Itchy skin is a common and debilitating side-effect of kidney disease and can affect up to 70% of people with stage 4 or 5 CKD. The causes are multifactorial, including calcium and phosphate imbalance, inadequate dialysis, overactive parathyroid gland activity, high levels of magnesium and vitamin A, and nerve changes in the skin.

Management

- Ensure that there are no other causes for pruritus (e.g., skin disease, scabies, inadequate dialysis, calcium/phosphate abnormalities).
- Avoid long hot showers.
- Application of ice.
- Evening primrose oil.
- Skin emollients.
- Avoid use of soaps/detergents.
- Topical capsaicin (may not be tolerated because of transient burning feeling on the skin).
- If both pruritus and restless legs is present, consider gabapentin.
- For persistent pruritus, consider referral to a dermatologist for ultraviolet light B (UVB) therapy or to investigate other causes of pruritis.
- Consider a selective kappa opioid receptor agonist (e.g., Difelikefalin) for treatment of moderate to severe pruritis in patients with CKD on haemodialysis.

Restless legs

Restless legs syndrome (RLS) is common in CKD. More than one quarter of people with CKD have RLS. It is more prevalent in people who receive dialysis, with almost one third having RLS.[108]

Management

- Evaluate severity using the Restless Legs Syndrome Rating Scale.[109]
- Check iron status and replace if deficient (absolute/relative).
- Home therapies such as massage, warm baths, warm/cool compresses, relaxation techniques, and exercise.
- Low dose dopaminergic agents or dopamine agonists.
- Pramipexole.

Sleep apnoea

Sleep apnoea can affect up to 50% of people with eGFR <15 mL/min/1.73m^2 and is a significant cause of refractory hypertension.

Management

- Weight reduction (see page 32).
- Avoid central nervous system depressants (including alcohol).
- Referral for assessment of severity of sleep apnoea.
- Continuous positive airway pressure (CPAP) therapy (if obstructive pattern).

Uraemia

Uraemia is a syndrome seen in stage 4 or 5 CKD. It is caused by the accumulation of the breakdown products of protein metabolism. The symptoms include anorexia, nausea, vomiting, lethargy, and in the advanced stages – confusion (encephalopathy), muscle twitching, pericarditis, fluid overload, convulsions, and coma. Although urea and creatinine are the substances measured, the symptoms are most likely due to the accumulation of other unmeasured toxic end products. By the time uraemia becomes symptomatic, dialysis is typically indicated.

Management

- Dialysis should be commenced based on assessment of uraemic symptoms, not eGFR or biochemistry.
- If non-dialysis pathway is planned, the patient should be reviewed by a specialist kidney supportive care team for assessment of symptoms and non-dialysis therapies. These may include dietary modifications, fluid restriction, anti-emetics, and therapy to address pruritus.

5

Progressive CKD

Indications for referral to a nephrologist[32, 110]

Appropriate referral is associated with positive outcomes, including:

- Reduced rate of progression to kidney failure.
- Decreased morbidity and mortality.
- Decreased need for and duration of hospitalisation.
- Increased likelihood of timely preparation of permanent dialysis access prior to dialysis onset.
- Increased likelihood of kidney transplantation.

Tests recommended prior to referral:

- Current blood chemistry and haematology
- uACR and urine microscopy for red cell morphology
- Current and historical blood pressure
- +/- Kidneys Ureters Bladder ultrasound scan

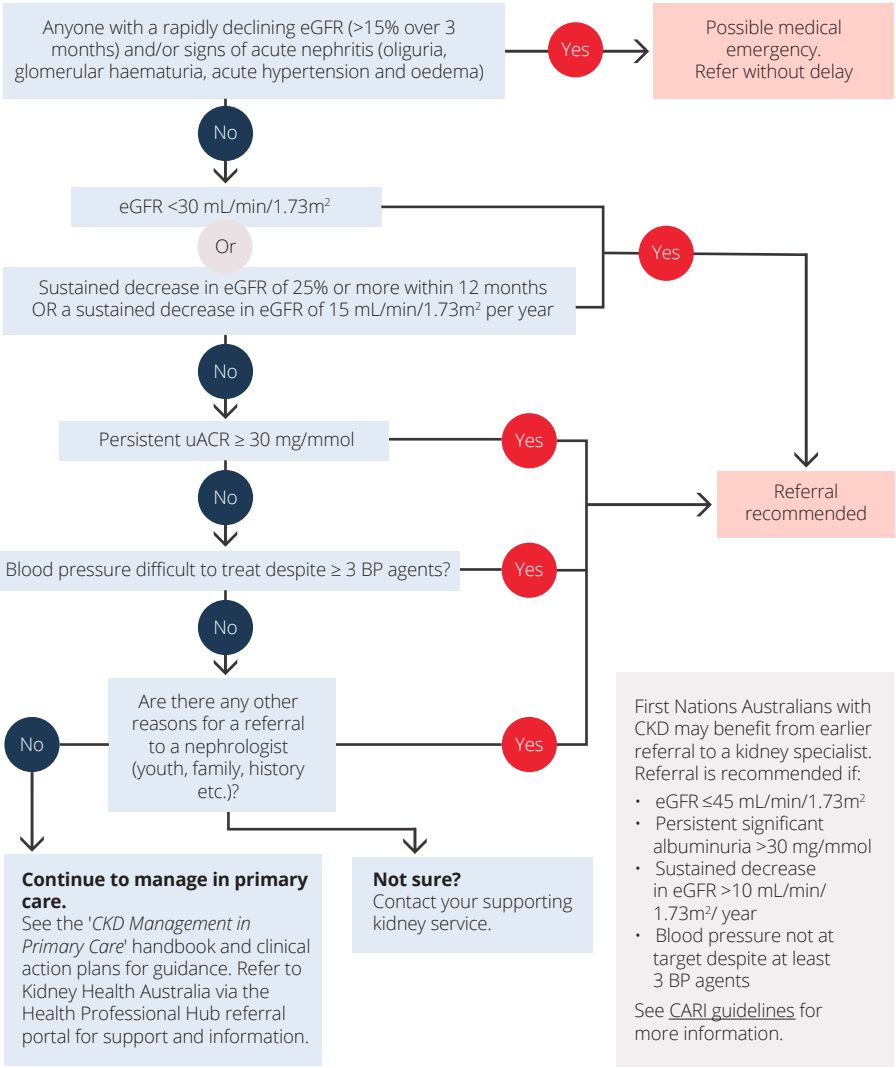
Clinical tip

Anyone with a rapidly declining eGFR (>5 mL/min/1.73m^2 over 3 months) and/or signs of acute nephritis (oliguria, haematuria, acute hypertension, and oedema) should be regarded as a medical emergency and referred without delay.

Further information and resources

- Kidney Health Australia Health Professional Hub: How to guide – Refer to Nephrology and Referral Letter Template.

Algorithm for appropriate referral to a nephrologist



* The decision to refer or not must always be individualised, particularly in younger individuals where the indications for referral may be less stringent. Discuss management issues with a specialist where it may not be necessary for the person with CKD to be seen by the nephrologist.

Where referral to a Nephrologist is not possible, as may be the case for people located in regional and remote areas, we recommend contacting your supporting kidney service to discuss options for referral, which may include telehealth consultations.

Advance care planning

- This can be a mix of actions that lead to planning towards the end of life.
- Advance care planning is distinct from dialysis treatment decision making and can occur whilst the person is still receiving treatment.
- Advance care planning should be initiated in:
  - All competent patients aged 65 years and above, and
  - All competent patients, irrespective of age, who fulfil one or more of the following criteria:
    - the treating clinician considers that existing medical conditions will reduce life expectancy.
    - significant comorbidities.
    - poor functional status.
    - chronic malnutrition.
    - poor Quality of Life.

Further information and resources

- Advance Care Planning Australia.
- Palliative Care Australia.

Treatment options for kidney failure

- There are three treatment options for kidney failure: dialysis which may be done at home or in a hospital or dialysis centre; kidney transplantation; and comprehensive conservative care.
- Patients should be referred to a nephrology service allowing adequate time for treatment options to be explored and considered prior to starting treatment.
- Patients and their families or carers should receive sufficient information and education regarding the nature of kidney failure and the options for the treatment to allow them to make an informed decision about the management of their condition.
- A shared decision-making approach between patients their families and healthcare team is recommended.

Further information and resources

- Kidney Health Australia booklet: Introduction to Kidney Disease Treatment Options.
- Kidney Health Australia decision aid for people affected by CKD and health professionals: My Kidneys My Choice.
- NSW Renal Network website: Renal Supportive Care.

Abbreviations

ACE inhibitor	Angiotensin-converting enzyme inhibitor
ACR	Albumin/creatinine ratio
ADPKD	Autosomal dominant polycystic kidney disease
AKI	Acute kidney injury
ABPM	Ambulatory blood pressure monitoring
ANCA	Antineutrophil cytoplasmic antibodies
Anti-GBM	Anti-glomerular basement membrane diseases
ANA	Antinuclear antibody
APD	Automated peritoneal dialysis
APNA	Australian Primary Healthcare Nurses Association
ARB	Angiotensin II receptor blocker
ARNI	Angiotensin receptor neprilysin inhibitor
BMD	Bone mineral density
BMI	Body mass index
BNP	Brain natriuretic peptide
BP	Blood pressure
BSA	Body surface area
BGL	Blood glucose level
CARI	Caring for Australasians with Renal Impairment
CIN	Contrast-induced nephropathy
CKD	Chronic kidney disease
CKD-EPI	Chronic Kidney Disease Epidemiology Collaboration
COVID-19	Coronavirus disease - 2019
Cox-2 inhibitors	Cyclooxygenase-2 inhibitors
CPAP	Continuous positive airway pressure
CrCl	Creatinine clearance
CVD	Cardiovascular disease
DASS-21	Depression Anxiety and Stress scale 21
DKD	Diabetic kidney disease
DPP-4 inhibitors	Dipeptidyl peptidase-4 inhibitors
eGFR	Estimated glomerular filtration rate
ENA	Extractable nuclear antigen
ESA	Erythropoiesis stimulating agent
ESR	Erythrocyte sedimentation rate
FLC	Free light chains
GFR	Glomerular filtration rate
GLP-1 RA	Glucagon-like peptide-1 receptor agonists
Hb	Haemoglobin

HBV	Hepatitis B virus
HCV	Hepatitis C virus
HCO_3	Bicarbonate
HDL	High-density lipoprotein
HF	Heart failure
HIV	Human immunodeficiency virus
HBPM	Home blood pressure monitoring
HR	Hazard ratio
hsCRP	High-sensitivity C-reactive protein
HSVD	Heart, stroke and vascular disease
IV	Intravenous
K^+	Potassium
K10	Kessler Psychological Distress Scale
KDIGO	Kidney Disease Improving Global Outcomes
KHA	Kidney Health Australia
KUB ultrasound	Kidney ureters bladder ultrasound
LDL	Low-density lipoprotein
MBD	Mineral and bone disorder
MBS	Medicare Benefit Schedule
MRA	Mineralocorticoid receptor antagonists
NHMRC	National Health and Medical Research Council
NIP	National Immunisation Program Schedule
NSAIDs	Non-steroidal anti-inflammatory drugs
PBS	Pharmaceutical benefits scheme
PCR	Protein: creatinine ratio
PD	Peritoneal dialysis
PKD	Polycystic kidney disease
PTH	Parathyroid hormone
RACGP	Royal Australian College of General Practitioners
RAS	Renin-angiotensin system
RLS	Restless legs syndrome
SGLT2 inhibitor	Sodium-glucose linked transporter-2 inhibitor
SNAP	Smoking, nutrition, alcohol, physical activity
SNRI	Selective serotonin and norepinephrine reuptake inhibitors
SSRI	Selective serotonin reuptake inhibitor
Trig	Triglycerides
TSAT	Transferrin saturation
uACR	Urine albumin/creatinine ratio
UTI	Urinary tract infection
UVB	Ultraviolet light B

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Index

Where to go for more information

Kidney Health Australia

At Kidney Health Australia, we're passionate about *Healthy Kidneys for all Australians*. As the recognised voice for CKD in Australia, we're driving awareness and earlier detection of CKD and championing best practice care to slow disease progression and improve health outcomes. We support people living with CKD, their healthcare teams, and the research community to achieve better outcomes. Through advocacy, programs and partnerships we're changing the paradigm from kidney failure to kidney preservation.

Resources for Health Professionals

We offer a range of education, practice tools and resources for health professionals working in primary care.

- Free accredited CPD education
- Quality improvement activities
- Dedicated Health Professional resources Hub
- Downloadable 'how to guides' and referral templates

Visit the Kidney Health Australia Health Professional Hub

Resources and services for patients:

The Kidney Health Australia team is here to support your patients living with CKD.

- Evidence-based factsheets, books and videos on CKD
- Tailored CKD education programs for patients
- Peer support programs
- Big Red Kidney Bus holiday dialysis service
- Kidney Helpline
- KidneyHealth4Youth support and events for young people with kidney disease.

Visit the Kidney Health Australia website

Kidney Helpline

Free health information service for anyone requiring assistance with managing their kidney health, understanding their kidney disease diagnosis or information on Kidney Health Australia support programs.

- Free call 1800 454 363
- Email kidney.helpline@kidney.org.au

Related resources

CARI Guidelines

cariguidelines.org

Provides a range of Australian evidence-based clinical practice guidelines for managing CKD and related conditions.

KDIGO Guidelines

kdigo.org

Provides a range of international evidence-based clinical practice guidelines for managing CKD.

Royal Australian College of General Practitioners (RACGP)

racgp.org.au

Provides a range of clinical guidelines, education and resources for GPs that are relevant to people with CKD.

Local HealthPathways

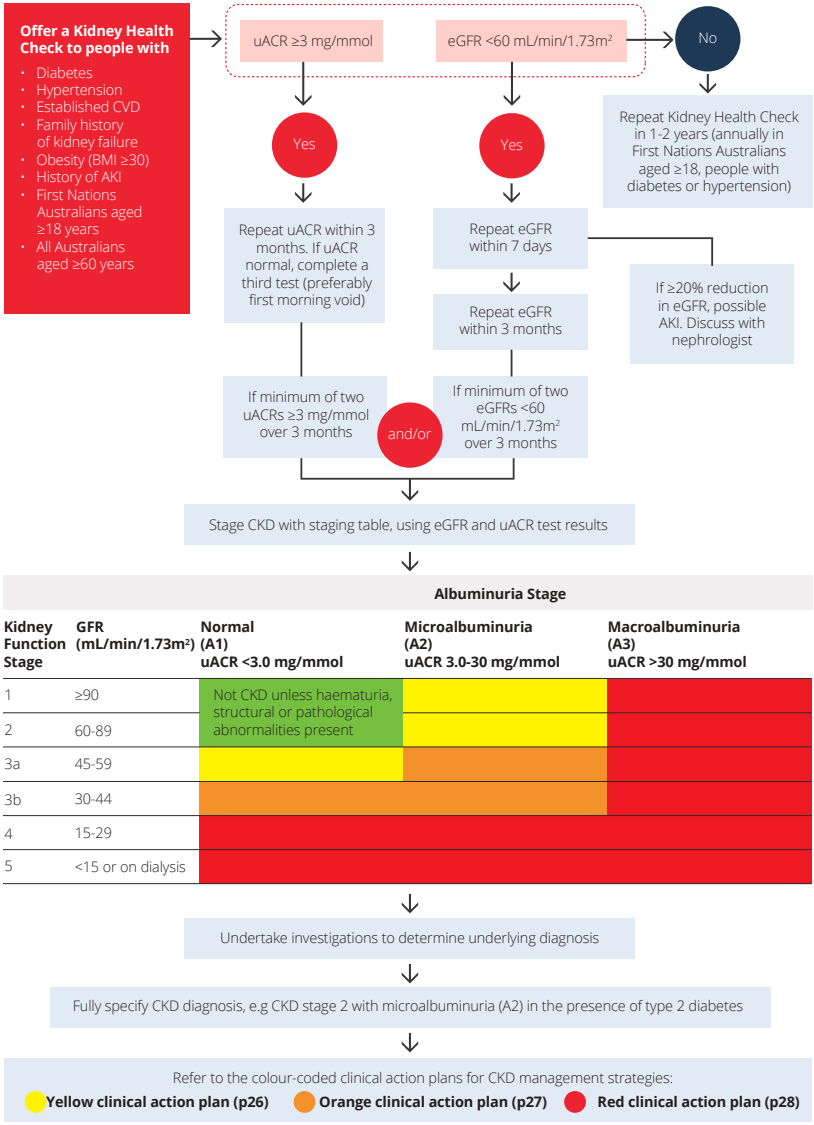
Most regions of Australia have web-based HealthPathways that provide evidence-based, localised advice on management and referral for a wide range of diseases and conditions, including CKD. Primary Health Networks can provide further details and access to their local Health Pathways.

Practice Software

Practice software tools such as PENCAT and Primary Sense can be a useful practice tool for identifying people in your practice at risk of or with CKD. Correct coding of CKD combined with the utilisation of medical software search functionality is useful in setting up quality improvement frameworks within practice.

Kidney Health Australia

Algorithm for initial detection and diagnosis of CKD



		Albuminuria Stage		
Kidney Function Stage	**GFR (mL/min/1.73m²)**	**Normal (A1) uACR <3.0 mg/mmol**	**Microalbuminuria (A2) uACR 3.0-30 mg/mmol**	**Macroalbuminuria (A3) uACR >30 mg/mmol**
1	≥90	Not CKD unless haematuria, structural or pathological abnormalities present		
2	60-89			
3a	45-59			
3b	30-44			
4	15-29			
5	<15 or on dialysis			

Undertake investigations to determine underlying diagnosis

Fully specify CKD diagnosis, e.g CKD stage 2 with microalbuminuria (A2) in the presence of type 2 diabetes

Refer to the colour-coded clinical action plans for CKD management strategies:

Yellow clinical action plan (p26) **Orange clinical action plan (p27)** **Red clinical action plan (p28)**