Chronic Kidney Disease (CKD) Management in General Practice

Kidney® Health Australia

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

3rd Edition 2015 • www.kcat.org.au

Prevent, Detect, Support.

KCAT

APNA CPD ENDORSED 2015

Accepted clinical resource

ANZSN

Connect with us
Freecall 1800 454 363
www.kidney.org.au

Key clinical tips

Management of early CKD includes steps to reduce cardiovascular disease risk. Recommend lifestyle changes and prescribe ACE Inhibitors or ARBs to lower blood pressure and slow the progression of albuminuria.

People with moderate or severe CKD, defined as persistently having a urine ACR >25 mg/mmol (males) or >35 mg/mmol (females) or eGFR <45 mL/min/1.73m^2, are considered to already be at the highest risk (>15% probability in five years) of a cardiovascular event, and therefore should not be assessed using the absolute cardiovascular risk tool. Failure to recognise the presence of moderate to severe CKD may lead to a serious under-estimation of CVD risk in that individual.

ACE inhibitors and ARBs cause a reversible reduction in glomerular blood flow and GFR can decline when treatment is initiated. Provided the reduction is less than 25% within two months of starting therapy, the ACE inhibitor or ARB should be continued. If the reduction in GFR is more than 25% below the baseline value, the ACE inhibitor or ARB should be ceased and consideration given to referral to a Nephrologist.

CKD in itself is not a diagnosis. Attempts should be made to identify the underlying cause of CKD.

If eGFR is < 60 mL/min/1.73 m^2, retest within 7 days and consider:

- *clinical situations where eGFR results may be unreliable and/or misleading*
- *acute kidney damage*

Anyone with rapidly declining eGFR and/or signs of acute nephritis (oliguria, haematuria, acute hypertension and oedema) should be regarded as a medical emergency and referred without delay.

The combination of ACE inhibitor (or ARB), diuretic and NSAID or COX-2 inhibitor (except low-dose aspirin) can result in acute kidney injury (the "triple whammy"), especially if volume-depleted or CKD present. Ensure individuals on blood pressure medication are aware of the need to discuss appropriate pain relief medication with a General Practitioner or Pharmacist.

ACE inhibitors and ARBs may be temporarily discontinued during acute illness, but should be recommenced when the condition stabilises.

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

Care of elderly patients with CKD requires an individualised approach to address comorbidities, together with variability in functional status, life expectancy and health priorities.

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

The Australian Kidney Foundation
Trading as Kidney Health Australia
ABN 37 008 464 426 | Charity No. CH 0614

How to use this booklet

This booklet has been specifically designed to be easy to use and interactive. The front/back cover can be removed and used as a quick reference guide. Relevant links to patient fact sheets, websites, and additional resources are interspersed throughout the booklet.

This booklet is available in hard copy and electronic soft copy (free download from www.kidney.org.au). The electronic copy contains interactive hyperlinks, and all tables, algorithms and figures are also available as individual downloads.

Resources for you

CKD education

Kidney Health Australia provides accredited education for health professionals through our Kidney Check Australia Taskforce (KCAT) program. Accredited (RACGP, ACRRM, ACN, APNA) face to face and online learning modules are available free of charge to Australian health professionals.

KCAT education sessions support the recommendations made in this booklet and will facilitate translating these recommendations into best practice detection and management of CKD in primary care.

If you would like to undertake some education related to the contents of this booklet, please visit www.kcat.org.au for further information.

CKD Management in General Practice App

CKD-Go! is a free web-based app that allows you to view a personalised CKD Clinical Action Plan based on an individual's eGFR and urine albumin creatinine ratio results. Smart-phone compatible, the app can be viewed and downloaded at www.kidney.org.au.

Resources for your patients

Kidney Health Australia has a suite of brochures, health fact sheets, publications and self-management resources that give precise, up to date health promotion and disease prevention messages. A range of translated resources is also available.

Recommended consumer resources for people with early stages of CKD:

- Fact sheet: All about chronic kidney disease
- Fact sheet: Looking after yourself with chronic kidney disease
- Fact sheet: eGFR
- Fact sheet: How to look after your kidneys
- Publication: Back on the Menu

Recommended consumer resources for people with later stages of CKD:

- Fact sheet: Common kidney disease symptoms and management options
- Fact sheet: Treatment options
- Publication: Living with Kidney Failure
- Publication: Back on the Menu

Visit www.kidney.org.au to download a pdf or request a hard copy.

Foreword

This third edition of Chronic Kidney Disease (CKD) Management in General Practice is the synthesis of the evolving evidence that the management of kidney disease matters. The Kidney Check Australia Task Force (KCAT) -now in its 13th year- has produced this book in the hope that practitioners will find the recommendations helpful in individuals at risk or with kidney disease and above all be inspired to identify kidney disease in their patients. I wish to acknowledge Professor David Johnson – Chair of KCAT for the last 9 years - who has provided strong and consistent leadership without which KCAT may well have faltered.

Three facts drive KCAT in its task. The outstanding fact, confirmed in the recent Australian Health Survey, is that evidence of kidney disease exists in 10% of Australian adults yet only one in ten of those with it are aware of that fact. Truly this is a silent and under-recognised condition. Increased recognition of kidney disease in high risk people is our top priority and this can only realistically happen in the general practice setting.

The second fact is that even early kidney disease is associated with increased morbidity and mortality and this can be impacted by using the clinical action plans outlined in this book. The kidney world is waiting on a specific fix or treatment for kidney disease, hopefully applicable to most people at risk of progression, but until that comes much can be done that is effective and affordable.

Thirdly, to put this in perspective there is building high-level evidence that the presence of CKD is a greater risk factor for cardiovascular disease than is diabetes. Kidney disease is not just another risk – it is a strong and independent risk factor that when identified and managed properly will contribute significantly to the striking and continuing fall in cardiovascular mortality in Australia.

Our only hope of reducing the burden of kidney disease is to better identify and manage individuals with this condition. Our hope is that this book, wholly evidence-based and presented in a summary, practical style, will add to the ability of general practitioners to take on this task.

I must thank Dr Marie Ludlow who again used her great skill in collating the evidence, drawing all the contributions together and writing this book whilst maintaining unfailing good humour and positivity. The kidney world is in her debt.

Associate Professor Tim Mathew
AM, MBBS, FRACP

National Medical Director
Kidney Health Australia

Contents

What's new?

The 3rd edition of CKD Management in General Practice contains new sections on management of ***acute kidney injury*** (see page 28), and ***kidney stones*** (see page 30) as a response to demand for information about these common conditions.

Australian statistics show that for every new individual treated with dialysis or transplant there is one who is not, with the majority of these being elderly individuals[1]. Additional sections on ***treatment for Stage 5*** CKD (including greater acknowledgement of the non dialysis supportive care pathway) (see page 24), ***advance care plans*** (see page 26), and ***CKD in the elderly*** (see page 27) provide important primary health care education on these issues.

Shared decision making is a concept that is gaining traction in Australian clinical practice, and a new section on page 26 provides guidance on this topic.

There have been no changes to the key recommendations regarding detection and management of CKD from the 2nd to the 3rd edition, with targeted early detection using the 3-step ***Kidney Health Check*** (eGFR, urine ACR, blood pressure) (see page 11) still best practice.

The publication of the Kidney Disease: Improving Global Outcomes (KDIGO) guideline on ***lipid management*** in CKD[2] recommended lipid lowering medications for many people with CKD, and removed the recommendation to use statins to achieve specified lipid targets[2]. The new guidance adopts a 'set and forget' approach whereby prescription of statin or statin/ezetimibe combination is based on age, eGFR level, and cardiovascular disease risk, irrespective of CKD stage (see page 41). Once statin therapy (or combination statin/ezetimibe) is initiated there is no evidence to support ongoing monitoring of lipid levels.

The new anticoagulants (apixaban, dabigatran, rivaroxaban) have also been added to the list of ***commonly prescribed drugs*** that may need to be reduced in dose or ceased in CKD, and additional prescribing information regarding non loop diuretics and loop diuretics has been added (see page 21).

New resources that support the CKD Management in General Practice book include a web-based app (CKD-GO!), downloadable care plan templates, and sample referral letters.

Visit www.kcat.org.au to view these resources.

Why worry about chronic kidney disease (CKD)?

CKD is defined as the occurrence of kidney damage and/or reduced kidney function that lasts for three months or more.

In Australia, CKD is:

Common

- Approximately 1.7 million Australians (1 in 10) aged 18 years and over have indicators of CKD such as reduced kidney function and/or albumin in the urine[3].
- Fewer than 10% of the people with CKD are aware they have this condition[4].
- This means over 1.5 million Australians are unaware they have indicators of CKD.

How much CKD in Australia?

1 in 1400 on dialysis or living with a transplant

Harmful

- Kidney and urinary tract diseases are the 9th leading cause of death in Australia, killing more people each year than breast cancer, prostate cancer and road deaths[5].
- CKD is a stronger risk factor for future coronary events and all-cause mortality than diabetes[6].

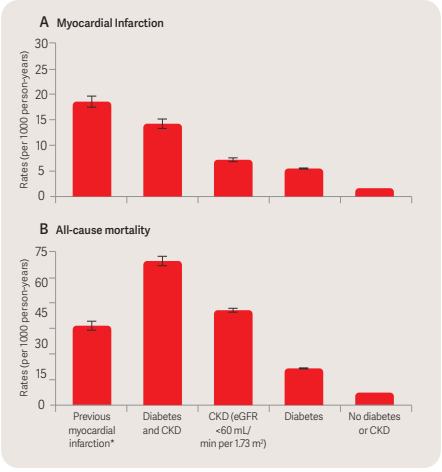
Treatable

- Early management of CKD (lifestyle changes, prescription of ACE inhibitors or ARBs) includes cardiovascular disease risk reduction.
- If CKD is detected early and managed appropriately, then the otherwise inevitable deterioration in kidney function can be reduced by as much as 50% and may even be reversible[7].

Clinical tip

Management of early CKD includes steps to reduce cardiovascular disease risk. Recommend lifestyle changes and prescribe ACE Inhibitors or ARBs to lower blood pressure and slow the progression of albuminuria.

Risk of coronary events and all-cause mortality according to the presence or absence of CKD, diabetes, and previous myocardial infarction [6]



Who is at risk of CKD?

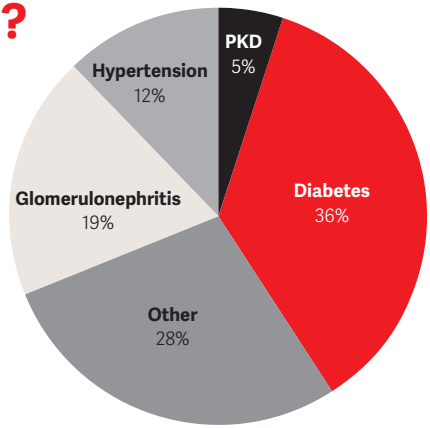
Adult Australians are at increased risk of developing CKD if they[8]:

- have diabetes
- have hypertension
- have established cardiovascular disease
- have a family history of kidney failure
- are obese (body mass index ≥ 30 kg/m^2)
- are a smoker
- are 60 years or older
- are of Aboriginal or Torres Strait Islander origin
- have a history of acute kidney injury (AKI)

What are the causes of end stage kidney disease (ESKD)?

The most common causes of ESKD in Australia are[9]:

- diabetic kidney disease
- glomerulonephritis
- hypertensive vascular disease
- polycystic kidney disease (PKD)



Clinical presentation of CKD

CKD is generally asymptomatic.

- Up to 90% of kidney function may be lost before symptoms are present, so annual checking of those at risk is essential.
- People with CKD may not notice any symptoms until they reach Stage 5 CKD (see Staging Table on page 19).

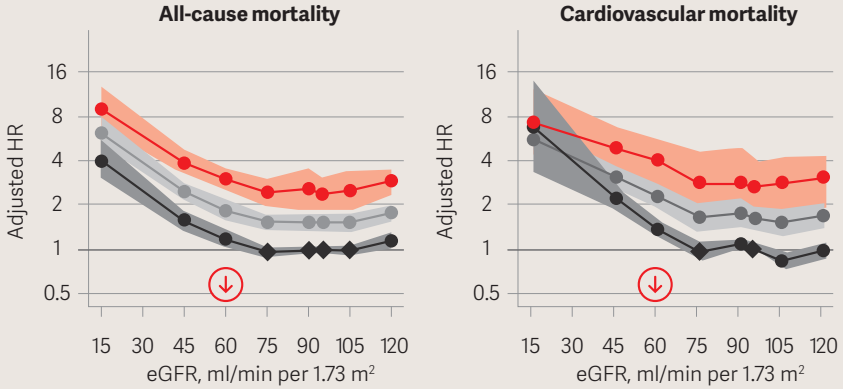
The first signs of CKD may be general, and include but are not limited to:

- hypertension
- pruritus
- nocturia
- restless legs
- haematuria
- dyspnoea
- lethargy
- nausea/vomiting
- malaise
- anorexia

CKD and cardiovascular disease

- CKD is a more important risk factor for cardiovascular disease than diabetes[6].
- Both reduced eGFR and significant albuminuria are independent risk factors for cardiovascular disease[10].
- Recent studies have confirmed that even early CKD constitutes a significant risk factor for cardiovascular events and death[11].
- 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[12].

Higher urinary albumin excretion increases relative risk of all-cause mortality and cardiovascular mortality at all levels of eGFR[10]



Black - normal albuminuria • **Grey** - microalbuminuria • **Red** - macroalbuminuria
HR = Hazard Ratio for mortality

Absolute cardiovascular risk assessment[13, 14]

- A comprehensive risk assessment, using an absolute risk approach, is recommended to assist general practitioners effectively manage their patient's cardiovascular risk by providing a meaningful and individualised risk level.
- Absolute risk is the numerical probability of an event occurring within a specified period, expressed as a percentage. For example, if your patient's risk is 15%, there is a 15% probability that they will experience a cardiovascular event within 5 years.

How to assess absolute cardiovascular risk

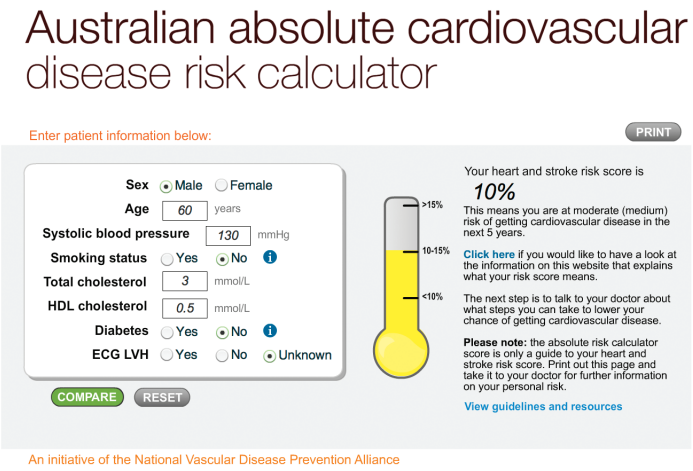
Who to target for risk assessment	All adults aged ≥45 years (or ≥ 35 years if of Aboriginal and Torres Strait Islander origin) *without* existing cardiovascular disease or other clinically determined high risk factor

Clinically determined high risk factors
Adults with any of these conditions are automatically at HIGH risk of cardiovascular disease
	• Moderate or severe CKD
	  • persistent urine ACR > 25 mg/mmol in males or > 35 mg/mmol in females
	  or
	  • eGFR < 45 mL/min/1.73m^2
	• Diabetes and age > 60 years
	• Diabetes with microalbuminuria
	  • persistent urine ACR > 2.5 mg/mmol in males or > 3.5 mg/mmol in females
	• Previous diagnosis of familial hypercholesterolaemia
	• Systolic blood pressure ≥ 180 mmHg or diastolic blood pressure ≥ 110 mmHg
	• Serum total cholesterol > 7.5 mmol/L
	• Aboriginal and Torres Strait Islander peoples aged > 74 years

How to perform risk assessment	Web calculator www.cvdcheck.org.au
What do results mean*	• *High:* greater than 15% risk of cardiovascular disease within next five years • *Moderate:* 10-15% risk of cardiovascular disease within next five years • *Low:* Less than 10% risk of cardiovascular disease within next five years

* Provide lifestyle and pharmacological management strategies (if indicated) based on the patient's risk level and clinical judgement (e.g., high risk require more intensive intervention and follow up).

Australian absolute cardiovascular disease risk calculator and associated health professional and patient resources are available at www.cvdcheck.org.au



Clinical tip

People with moderate or severe CKD, defined as persistently having a urine ACR >25 mg/mmol (males) or >35 mg/mmol (females) or eGFR <45 mL/min/1.73m^2, are considered to already be at the highest risk (>15% probability in five years) of a cardiovascular event, and therefore should not be assessed using the absolute cardiovascular risk tool. Failure to recognise the presence of moderate to severe CKD may lead to a serious under-estimation of CVD risk in that individual.

Reducing cardiovascular risk - lifestyle modification[13]

- People at all cardiovascular risk levels can make improvements to their health and reduce their risk of cardiovascular disease by making lifestyle changes.
- See the table on page 53 for guidance on basic lifestyle advice. For more detailed advice refer to the relevant guidelines.

Reducing cardiovascular risk - pharmacotherapy[13]

- CKD can cause and aggravate hypertension, and hypertension can contribute to the progression of CKD.
- Reducing blood pressure to below target levels is one of the most important goals in management of CKD (see blood pressure targets on page 39).
- In people with CKD, blood pressure lowering therapy should begin with either ACE inhibitor or ARB.
  - Combined therapy of ACE inhibitor and ARB is not recommended.
  - Maximal tolerated dose of ACE inhibitor or ARB is recommended.
- Hypertension may be difficult to control and multiple (3 - 4) medications are frequently required.
- Assess risk of atherosclerotic events and consider treating with an anti-platelet agent unless there is an increased bleeding risk[15].
- See page 39 for more information regarding management of hypertension in people with CKD.

Consumer fact sheets 'Cardiovascular disease and chronic kidney disease' and 'Blood pressure and chronic kidney disease' are available to download at www.kidney.org.au

Clinical tip

ACE inhibitors and ARBs cause a reversible reduction in glomerular blood flow and GFR can decline when treatment is initiated. Provided the reduction is less than 25% within two months of starting therapy, the ACE inhibitor or ARB should be continued. If the reduction in GFR is more than 25% below the baseline value, the ACE inhibitor or ARB should be ceased and consideration given to referral to a Nephrologist.

Early detection of CKD

- Increasing amounts of albumin in the urine correlate directly with an increased rate of progression to ESKD, and increased cardiovascular risk.
- eGFR correlates well with complications of CKD and an increased risk of adverse outcomes such as cardiovascular morbidity and mortality.
- Early intervention with blood pressure reduction and use of ACE inhibitors or ARBs can reduce progression and cardiovascular risk by up to 50%, and may also improve quality of life.
- Testing for CKD should not be universal, but should be targeted and performed in individuals at increased risk of developing CKD[16].
- Serum creatinine is an insensitive marker for detecting mild to moderate kidney disease – eGFR is the preferred test[17].
- 50% or more of kidney function can be lost before the serum creatinine rises above the upper limit of normal.

Early detection of CKD using Kidney Health Check[18, 19]

Indications for assessment*	Recommended assessments	Frequency
Diabetes Hypertension Established cardiovascular disease** Family history of kidney failure Obesity (BMI ≥30 kg/m^2) Smoker Aboriginal or Torres Strait Islander origin aged ≥ 30 years¶	Urine ACR, eGFR, blood pressure If urine ACR positive repeat twice over 3 months (preferably first morning void). If eGFR < 60mL/min/1.73m^2 repeat within 7 days.	Every 1-2 years§
History of acute kidney injury	See recommendations on page 28	

* Whilst being aged 60 years of age or over is considered to be a risk factor for CKD, in the absence of other risk factors it is not necessary to routinely assess these individuals for kidney disease.

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

§ Annually for individuals with diabetes or hypertension.

¶ See page 12 for more detail regarding recommendations for testing in Aboriginal and Torres Strait Islander peoples.

Aboriginal and Torres Strait Islander peoples

Latest data from the Australian Aboriginal and Torres Strait Islander Health Survey[20] showed:

- Age-standardised incidence of Stage 5 CKD is significantly higher in Aboriginal and Torres Strait Islander peoples compared with non Aboriginal and Torres Strait Islander peoples.
- Indigenous Australians are twice as likely to have signs of CKD, and four times as likely to have Stages 4-5 CKD, than non-Indigenous Australians.
- 90% of Aboriginal and Torres Strait Islanders with CKD are not aware that they have this condition.

Recommendations for CKD detection in Aboriginal and Torres Strait Islander peoples[19]

Indications for assessment*	Recommended assessments	Frequency
People 18-29 years without any CKD risk factors	Screen for CKD risk factors (overweight or obesity, diabetes, elevated blood pressure, smoking, family history of kidney disease)	As part of annual health assessment
People 18-29 years with one of the following CKD risk factors: • Family history of CKD or premature CVD • Overweight/obesity • Smoking • Diabetes • Elevated blood pressure All people ≥30 years	Urine ACR, eGFR, blood pressure If urine ACR positive repeat twice over 3 months (preferably first morning void). If eGFR < 60mL/min/1.73m^2 repeat within 7 days.	Every two years (or more frequently if CVD risk is elevated)

For further detailed information refer to the National Guide to a Preventive Health Assessment for Aboriginal and Torres Strait Islander People[19] (www.naccho.org.au)

Benefits of identifying Aboriginal and Torres Strait Islander peoples:

- awareness of increased risk of CKD and cardiovascular disease and importance of screening other family members for CKD
- able to access annual health check (Medicare item 715)
- eligible for Aboriginal and Torres Strait Islander peoples-specific pharmaceutical benefits
- may be eligible for "Closing the Gap" scheme

Definition of CKD

CKD is defined as:

- an estimated or measured glomerular filtration rate (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, irrespective of the underlying cause:
  - albuminuria
  - haematuria after exclusion of urological causes
  - structural abnormalities (e.g., on kidney imaging tests)
  - pathological abnormalities (e.g., renal biopsy)

Three components to a diagnosis of CKD

CKD Stage	with...	due to...
1/2/3a/3b/4/5	normoalbuminuria or microalbuminuria or macroalbuminuria	presumed/ confirmed pathology
eGFR	Urine ACR	Various recommended tests
See page 15	See page 18	See page 14

Clinical tip

CKD in itself is not a diagnosis. Attempts should be made to identify the underlying cause of CKD.

The following diagnostic evaluation tests for CKD are always indicated[8]:

- Renal ultrasound scan
- Repeat (within 1 week) serum urea/electrolytes/creatinine/eGFR/albumin. If eGFR continues to decrease refer to acute kidney injury management plan (see page 28)
- Full blood count, CRP, ESR
- Urine ACR (preferably on a first morning void to minimise postural effect on albumin excretion, although a random urine is acceptable)
- Fasting lipids and glucose
- Urine microscopy for dysmorphic red cells, red cell casts or crystals

The following diagnostic evaluation tests for CKD are sometimes indicated[8]:

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)	Anti-glomerular basement membrane antibody Anti-neutrophil cytoplasmic antibody Anti-nuclear antibodies Extractable nuclear antigens Complement studies
Risk factors for HBV, HCV or HIV (these conditions are associated with an increased risk of glomerular disease)	HBV, HCV, HIV serology
Age > 40 years and possible myeloma is suspected	Serum and urine protein electrophoresis

Tests used to investigate CKD

Glomerular Filtration Rate (GFR)[17]

- GFR is accepted as the best overall measure of kidney function.
- eGFR is a more sensitive marker for CKD than serum creatinine alone.
- 50% or more of kidney function can be lost before the serum creatinine rises above the upper limit of normal.
- Normal serum creatinine measurements do not exclude serious loss of kidney function.
- GFR can be estimated (eGFR) from serum creatinine using prediction equations.

How to assess eGFR[17]

- eGFR is automatically reported (using the CKD-EPI equation) with requests for serum creatinine in individuals aged ≥ 18 years.
- The CKD-EPI equation has been shown to have greater accuracy and precision for eGFR when compared to the Modification of Diet in Renal Disease (MDRD) and Cockcroft-Gault formulae.
- Further investigation of reduced eGFR is only required if the eGFR is < 60 mL/min/1.73 m^2.

Clinical tip

If eGFR is < 60 mL/min/1.73 m^2, retest within 7 days and consider:

- *clinical situations where eGFR results may be unreliable and/or misleading*
- *acute kidney damage*

Clinical situations where eGFR results may be unreliable and/or misleading[21]

- Acute changes in kidney function (e.g., acute kidney injury)
- 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
- Diseases of skeletal muscle, paraplegia, or amputees (may overestimate eGFR)
- High muscle mass (may underestimate eGFR)
- Children 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., fenofibrate, trimethoprim)
- Pregnancy (see below)

eGFR and drug dosing[17]

- Dose reduction of some drugs is recommended for people with reduced kidney function (see page 21).
- Manufacturers' renal dosing recommendations for medications are often based on Cockcroft-Gault estimates of creatinine clearance (CrCl mL/min).
- However, eGFR provides a valid estimate of renal 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 drugs with a narrow therapeutic index, therapeutic drug monitoring or a valid marker of drug effect should be used to individualise dosing.
- 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).
- To revert to an uncorrected eGFR:

$$\text{CKD-EPI eGFR result in mL/min/1.73m}^2 \times \frac{\text{Individual's BSA}}{1.73} = \text{eGFR result in mL/min}$$

Where BSA = 0.007184 x Weight in kg $^{0.425}$ x Height in cm $^{0.725}$ (Du Bois formula)

Use of eGFR in various ethnic populations

- The CKD-EPI formula has been validated as a tool to estimate GFR in some non-Caucasian populations, including Aboriginal and Torres Strait Islander people[22], and South-East Asian, African, Indian and Chinese individuals living in Western countries[23].

eGFR and pregnancy[17]

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

Consumer fact sheet 'eGFR – estimated glomerular filtration rate' is available to download at www.kidney.org.au

Urine albumin creatinine ratio (ACR)[16]

- Excessive amounts of proteins in the urine are a key marker of kidney damage and of increased renal and cardiovascular disease risk.
- 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.
- Urine ACR accurately predicts renal and cardiovascular risks in population studies.
- Reduction in urine ACR predicts renoprotective benefit in intervention trials.
- Elevated urine ACR is a more common sign of CKD than a decreased eGFR. In the latest Australian Health Survey, 8% of adults had abnormal urine ACR, while 4% had an abnormal eGFR result[3].

How to detect albuminuria[16]

- The preferred method for assessment of albuminuria in both diabetes and non-diabetes is urinary ACR measurement in a first morning void spot specimen.
- Urinary protein excretion follows a circadian pattern and tends to be highest in the afternoon, so ACR tests are most accurate when performed on early morning (first-void)[24].
- Where a first void specimen is not possible or practical, a random spot urine specimen for urine ACR is acceptable.
- A positive ACR test should be repeated on a first void sample to confirm persistence of albuminuria.
- Albuminuria is said to be present if at least two out of three ACR results are positive. CKD is present if the albuminuria is persistent for at least three months.
- Dipstick for protein in the urine is now no longer recommended as the sensitivity and specificity are not optimal.
- Urine ACR exhibits greater sensitivity than protein:creatinine ratio (PCR) for detecting lower amounts of clinically important albuminuria.

Factors other than CKD known to increase urine albumin excretion[16]

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

Consumer fact sheet 'Albuminuria' is available to download at www.kidney.org.au

Algorithm for initial detection of CKD

Offer Kidney Health Check to people with any of the following indications:

- Diabetes
- Hypertension
- Established cardiovascular disease
- Family history of kidney failure
- Obesity
- Smoking
- Aboriginal or Torres Strait Islander origin aged ≥ 30 years

Indication not present → Kidney Health Check not recommended

If urine ACR and eGFR are normal repeat Kidney Health Check in 1-2 years (annually if diabetes or hypertension present)

eGFR

Urine ACR

eGFR < 60 mL/min/1.73m²

Elevate urine ACR (males ≥2.5mg/mmol, females ≥3.5 mg/mmol)

≥ 20% reduction in eGFR → Possible acute kidney injury - discuss with Nephrologist

Repeat eGFR within 7 days

Repeat urine ACR twice within next 3 months (preferably first morning void)

Stable reduced eGFR

Repeat eGFR twice within 3 months

Minimum 2 out of 3 elevated urine ACR's present for ≥3 months

Minimum 3 reduced eGFR's present for ≥3 months

		Albuminuria Stage		
Kidney Function Stage	**GFR (mL/min/1.73m²)**	**Normal (urine ACR mg/mmol) Male: < 2.5 Female: < 3.5**	**Microalbuminuria (urine ACR mg/mmol) Male: 2.5-25 Female: 3.5-35**	**Macroalbuminuria (urine ACR mg/mmol) Male: > 25 Female: > 35**
1	<90	Not CKD unless haematuria, structural or pathalogical abnormalities present		
2	60-89			
3a	45-59			
3b	30-44			
4	15-29			
5	<15 or on dialysis			

Investigations to determine underlying diagnosis

Combine eGFR stage (1-5), albuminuria stage and underlying diagnosis to fully specify CKD (e.g., stage 2 CKD with microalbuminuria due to diabetic kidney disease).

Refer to colour-coded action plans for management strategies

Indications for referral to a Nephrologist[8, 25]

Appropriate referral is associated with:

- reduced rates of progression to ESKD
- decreased patient 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

Referral to a specialist renal service or Nephrologist is recommended in the following situations:

- eGFR < 30 mL/min/1.73m^2 (Stage 4 or 5 CKD of any cause)
- Persistent significant albuminuria (urine ACR ≥30 mg/mmol)
- A sustained decrease in eGFR of 25% or more OR a sustained decrease in eGFR of 15 mL/min/1.73m^2 within 12 months
- CKD with hypertension that is hard to get to target despite at least three anti-hypertensive agents

The individual's wishes and comorbidities should be taken into account when considering referral.

In the absence of other referral indicators, referral is not necessary if:

- Stable eGFR ≥30 mL/min/1.73m^2
- Urine ACR < 30 mg/mmol (with no haematuria)
- Controlled blood pressure

The decision to refer or not must always be individualised, and particularly in younger individuals the indications for referral may be less stringent. 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.

Recommended tests prior to referral:

- Current blood chemistry and haematology
- Urine ACR and urine microscopy for red cell morphology and casts
- Current and historical blood pressure
- Urinary tract ultrasound

Tests not recommended prior to referral:

- Urine culture
- Spiral CT angiogram for hypertension (without specialty advice)

For a sample referral letter template, visit www.kcat.org.au.

Clinical tip

Anyone with rapidly declining eGFR and/or signs of acute nephritis (oliguria, haematuria, acute hypertension and oedema) should be regarded as a medical emergency and referred without delay.

Medications

- It is important to review renally excreted medications, as well as avoid nephrotoxic medications in people with CKD.
- Dosage reduction or cessation of renally excreted medications 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/Pharmacist collaboration and are funded by Medicare item numbers.

Commonly prescribed drugs that may need to be reduced in dose or ceased in CKD include, but are not limited to:

Acarbose	Fenofibrate	Metformin*
Antivirals	Gabapentin	Opioid analgesics
Apixaban	Glibenclamide	Rivaroxaban
Benzodiazepines	Gliclazide	Saxagliptin
Colchicine	Glimeprimide	Sitagliptin
Dabigatran	Glipizide	Sotalol
Digoxin	Insulin	Spironolactone
Exanatide	Lithium	Valaciclovir
		Vildagliptin

* Metformin should be used with caution if GFR 30-60 mL/min/1.73m^2, and is not recommended if GFR < 30 mL/min/1.73m^2. It should be temporarily interrupted during periods of ill health and/or change in kidney function.

Commonly prescribed drugs that can adversely affect kidney function in CKD:

- Aminoglycosides
- Calcineurin inhibitors
- Gadolinium
- Lithium
- NSAIDs and COX-2 inhibitors - beware the 'triple whammy' (See Clinical tip)
- Radiographic contrast agents

Clinical tip

The combination of ACE inhibitor (or ARB), diuretic and NSAID or COX-2 inhibitor (except low-dose aspirin) can result in acute kidney injury (the "triple whammy"), especially if volume-depleted or CKD present. Ensure individuals on blood pressure medication are aware of the need to discuss appropriate pain relief medication with a General Practitioner or Pharmacist.

Managing hypertension medications in people with CKD

- ACE inhibitors or ARBs are an essential part of the best care approach for many patients in all stages of CKD.
- They cause a reduction in glomerular blood flow, and GFR can decline when treatment is initiated.
- Providing the reduction is less than 25% within two months of starting therapy, the ACE inhibitor or ARB should be continued.
- If the reduction in GFR is more than 25% below the baseline value, the ACE inhibitor or ARB should be ceased and consideration given to referral to a Nephrologist.
- Combined therapy with ACE inhibitor and ARB should be avoided except with specialist advice.
- Caution should be exercised if baseline K+ is ≥5.5 mmol/L, as rises in serum K+ of approximately 0.5 mmol/L are expected (see page 38).
- ACE inhibitors and ARBs can safely be prescribed at all stages of CKD and should not be deliberately avoided just because GFR is reduced.
- Both non-loop diuretics (e.g., thiazides) and loop diuretics (e.g., frusemide) are effective in all stages of CKD as adjunct antihypertensive therapy.
- Frusemide can be used safely for management of fluid overload 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/day is required, the efficacy is improved by dividing the daily dose.
  - The dose may need frequent adjustment, and is best guided by tracking the fluid status and the daily weight at home with the instructions to the patient being to use "as little frusemide as needed to control the swelling".
- Beta-blockers may be useful in people with coronary heart disease, tachyarrhythmias and heart failure, but are contraindicated in asthma and heart block.
- Calcium channel blockers may be used for people with angina, the elderly and those with systolic hypertension.

Clinical tip

ACE inhibitors and ARBs may be temporarily discontinued during acute illness, but should be recommenced when the condition stabilises.

Other medication resources for people with CKD:

- Appendix 1 from the "Australian Diabetes Society Position Statement on A New Blood Glucose Management Algorithm for Type 2 Diabetes"[26] for a list of medication options for people with diabetes and CKD www.mja.com.au/sites/default/files/issues/201_11/gun01187_Appendix1.pdf
- "A practical approach to the treatment of depression in patients with chronic kidney disease and end-stage renal disease" for a list of the most common classes of antidepressant medications with suggested dosing in kidney impairment, and potential adverse effects www.nature.com/ki/journal/v81/n3/fig_tab/ki2011358t2.html[27])
- Australian resource focusing on drug therapy in people with CKD www.renaldrugreference.com.au

Nutrition[8]

- People with CKD should be encouraged to eat a balanced and adequate diet according to energy requirements in line with the Dietary Guidelines of Australian Adults recommended by NMHRC.
- Australian guidelines recommend that people with eGFR < 30 mL/min/1.73m^2 should have individualised diet intervention involving an Accredited Practising Dietitian.
- Overweight or obese people with CKD should be prescribed caloric restriction under the management of an Accredited Practising Dietitian.

Nutrition targets for people with CKD and eGFR ≥ 30mL/min/1.73m^2[8]*

Parameter	Target
Protein	0.75-1.0 g/kg/day (no restriction necessary)
Salt	No greater than 100 mmol/day (or 2.3 g sodium or 6 g salt per day) Avoid adding salt during cooking or at the table Avoid salt substitutes that contain high amounts of potassium salts
Phosphate	No restriction necessary
Potassium	If persistent hyperkalaemia is present, consult Accredited Practising Dietitian regarding restricting intake and avoiding foodstuffs high in potassium
Fluid	Drink water to satisfy thirst Increased fluid intake is not necessary
Carbonated beverages	Avoidance is preferable Minimise intake to less than 250 mL per day

* People with eGFR < 30 mL/min/1.73m^2 should have nutrition targets set by an Accredited Practising Dietitian

Consumer fact sheet 'Nutrition and kidney disease' available to download at www.kidney.org.au

Treatment options for Stage 5 CKD

- Patients and their families or carers should receive sufficient information and education regarding the nature of Stage 5 CKD, and the options for the treatment to allow them to make an informed decision about the management of their condition.
- Treatment choice has more effect on lifestyle than it does on mortality or morbidity.
- A shared decision making approach is highly recommended.
- This is best supported by a decision aid, such as the My Kidneys My Choice Decision Aid, available at www.homedialysis.org.au/choosing/my-decision

Brief comparison of treatment options

Treatment	Types	Involves	Lifestyle impact/outcomes
Transplant	Living donor Deceased donor	• Surgery • Lifetime imunosuppressants • May wait 3-7 years for a deceased donor • Compatible live donor	• Freedom to work and travel once kidney function stabilised • Need to maintain a healthy diet, but no other restrictions • Survival rates good • Higher infections and cancer rate
Home Peritoneal Dialysis (PD)	Continuous Ambulatory Peritoneal Dialysis (CAPD)	Four daytime bags changed manually	• Need PD catheter • Simple, gentle and portable • 1 week training • Freedom to work and travel • Good quality of life • Usually lasts 2-5 years
	Automated Peritoneal Dialysis (APD)	Overnight exchanges managed by a machine	• As above with no requirement to change bags during the day
Home Haemodialysis	Daytime, 3-5 treatments weekly, 4-6 hrs duration	• Blood cleansed by artificial filter. • Surgery for fistula at least at least 3 months prior to use	• Average of 3 months for training • Flexible daily routine
	Night-time, 3-5 nights per week, 8 hrs duration		• As above, with more hours of dialysis offering better health outcomes

Brief comparison of treatment options cont.

Treatment	Types	Involves	Lifestyle impact/outcomes
Centre Based Haemodialysis	• Hospital or satellite centre • 3 x weekly • 4-6 hrs (individualised) • Occasional clinics offer overnight	• As above	• Strict routine • Strict diet • Transport to hospital or satellite centre needed • No training required • Infection risk
Non Dialysis Supportive Care	• No dialysis or transplant • Managed in the community • Supported by palliative care	• Medication and diet control • Advance care planning	• In most people, life expectancy will be decreased compared with dialysis or transplant • Dialysis therapy may not be associated with a survival advantage compared with non dialysis supportive care in elderly patients with two or more comorbidities

Shared decision making[28]

- Enables the clinician and patient to participate jointly in making an informed health decision.
- Involves discussing the options and their benefits and harms, and considering the patient's values, preferences and circumstances.
- Is not a one-off discussion, but an ongoing process that can be used to guide decisions about screening, investigations and treatments.
- Benefits include:
  - acknowledges patient values and preferences
  - enhances patient engagement
  - improves patient knowledge
  - supports evidence based care
- Although shared decision making can occur without tools, various decision support tools now exist. For more information visit www.safetyandquality.gov.au/our-work/shared-decision-making/

Five questions that clinicians can use to guide shared decision making[28]:

1. What will happen if we watch and wait?
2. What are your test or treatment options?
3. What are the benefits and harms of these options?
4. How do the benefits and harms weigh up for you?
5. Do you have enough information to make a choice?

Advance care plans

- This can be a mix of any actions that leads to planning towards the end of life.
- Advance care planning is distinct from dialysis treatment decision making, and can occur whilst treatment is still 'active'.
- 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
    - two or more significant comorbidities
    - poor functional status
    - chronic malnutrition
    - poor quality of life
- Visit www.advancecareplanning.org.au for information and resources.

Special issues in the elderly

- Most elderly people with CKD are asymptomatic.
- Relying on creatinine alone causes under-recognition of CKD.
- eGFR (which is adjusted for age) improves diagnostic accuracy.

Clinical tip

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

Appropriate referral

- Elderly patients with a stable eGFR ≥ 30 mL/min/1.73m^2, microalbuminuria, and controlled blood pressure can be managed successfully in primary care.
- 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.

Manage cardiovascular risk

- In people with CKD, death from cardiovascular disease is more common than ESKD at all ages.
- Manage cardiovascular risk (see page 8) using lifestyle and pharmacological management strategies (if indicated) based on the patient's risk level and clinical judgement.
- The goal of treatment is to improve the patient's functional capacity and quality of life, and to prevent injury from falls (e.g., postural hypotension, polypharmacy), rather than to achieve a target BP.

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 General Practitioner/ Pharmacist collaboration and are funded by Medicare item numbers.

Shared decision making

- Treatment choice has more effect on lifestyle than it does on mortality or morbidity.
- Dialysis therapy may not be associated with a survival advantage compared with non dialysis supportive care in elderly patients with two or more comorbidities.
- Utilise decision aid tools such as the My Kidneys My Choice Decision Aid, available at www.homedialysis.org.au/choosing/my-decision

Clinical tip

Care of elderly patients with CKD requires an individualised approach to address comorbidities, together with variability in functional status, life expectancy and health priorities.

Acute kidney injury (AKI)[29,30]

- AKI is a common syndrome, especially in hospitalised patients, and is independently and strongly associated with increased morbidity and mortality.
- AKI is diagnosed either by detection of a sudden increase in serum creatinine, OR with persistent oliguria (see below).

Risk factors for AKI

Pre-existing risk factors	Modifiable kidney insults
CKD	Hypovolaemia
Other chronic disease	Sepsis
Diabetes	Critical illness
Heart/lung/liver disease	Circulatory shock
Cancer	Burns
Anaemia	Trauma
Advanced age	Drugs (e.g., triple whammy)
Female gender	Radiocontrast agents
	Poisonous plants and animals (e.g., snakes, spiders)

- CKD increases the risk of AKI, and an episode of AKI in turn increases the likelihood of subsequent development of CKD, highlighting the need for ongoing surveillance.
- General practice is in a unique position to identify people at increased of AKI and address potentially modifiable exposures to prevent the occurrence of AKI.

AKI management plan

How to prevent AKI	• Identify all CKD 3-5 patients as increased risk for AKI • Early identification of patients at risk with acute illness, and consider temporary cessation of ACE Inhibitor/ARB/diuretics with hypovolaemia/hypotension • Minimise and monitor NSAIDs with CKD
How to diagnose AKI	• Increase in serum creatinine ≥ 25 µmol/l within 48 hours; or • Increase in serum creatinine to ≥ 1.5 times baseline, which is known or presumed to have occurred within the prior 7 days; or • Significant reduction in urine output compared with normal output
What to do during an AKI episode	• Remove risks in early stage of illness • Seek specialist advice early • Systematic fluid assessment and medication review for all patients at risk when acute illness occurs
What to do after an AKI episode	• Follow-up within 30 days after discharge, and then GP or Nephrology follow-up as required. • Annual Kidney Health Check for subsequent 3 years • Self-management to monitor and reduce risk of subsequent exposures

Kidney stones[31]

- 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, family history and Indigenous status.
- 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 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

- Existing calcium stones typically 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 to achieve this.
- 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 2L 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 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:
  - thiazides to reduce calcium excretion
  - allopurinol to reduce hyperuricosuria
  - citrate for hypocitraturia

Acute management

- The acute management of a stone episode is usually performed 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 (<5mm) 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 comply with a regimen that is devised after initial evaluation of the stone type and the risk factors present in the individual.

Consumer fact sheet 'Kidney stones' available to download at www.kidney.org.au

Multidisciplinary care

The management of CKD is always a collaborative effort, involving at least the individual and their General Practitioner. As kidney function declines, and as complications and comorbidities increase, it is likely that the contribution of others will be needed for optimal care.

The efficient integration of their various contributions becomes more challenging as the number of health professionals involved in the individual's care increases. The General Practitioner plays a crucial role, sustaining an ongoing relationship with the patient and their family, coordinating the care provided by others and ensuring that this care remains focused on the person's own goals and priorities.

At times the General Practitioner may be required to advocate for the patient with other professionals. In addition, he or she has continuing responsibility for the patient's primary care, which may include:

- supporting and assisting the patient in the management of their kidney disease and other chronic health problems
- responding appropriately to new symptoms
- screening for developing problems and comorbidities
- provision of health promotion and disease prevention advice and interventions
- providing appropriate vaccinations
- assistance with addressing psychosocial issues

Even if the patient progresses to Stage 5 CKD and has regular contact with the dialysis or transplant team, the General Practitioner, practice nurse, practice staff and other primary healthcare professionals remain vital to optimal care.

In Australia, a number of Medicare items are designed to support proactive, integrated, and multidisciplinary care for people with chronic disease. More information can be found at www.health.gov.au/mbsprimarycareitems.

Yellow clinical action plan

eGFR ≥60 mL/min/1.73m² with microalbuminuria or
eGFR 45-59 mL/min/1.73m² with normoalbuminuria

Goals of management

- Investigations to determine underlying cause
- Reduce progression of kidney disease
- Assessment of Absolute Cardiovascular Risk
- Avoidance of nephrotoxic medications or volume depletion

Management strategies

Frequency of review

- Every 12 months

Clinical assessment

- blood pressure
- weight

Laboratory assessment

- urine ACR (see page 18)
- eGFR (see page 15)
- biochemical profile including urea, creatinine and electrolytes
- HbA1c (for people with diabetes)
- fasting lipids

Other assessments

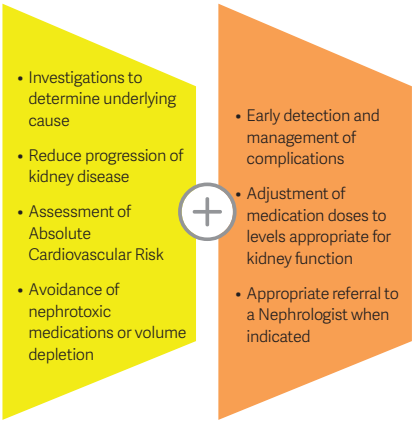
- assess absolute cardiovascular risk (see page 8)
- blood pressure reduction (see page 10)
- lifestyle modification (see page 10)
- lipid lowering treatment (where appropriate for risk factor reduction) (see page 41)
- glycaemic control (see page 37)
- avoid nephrotoxic medication or volume depletion (see page 21)
- multidisciplinary care (see page 31)

Care Plan Template available to download at www.kcat.org.au

Orange clinical action plan

eGFR 30-59 mL/min/1.73m² with microalbuminuria or
eGFR 30-44 mL/min/1.73m² with normoalbuminuria

Goals of management



Management strategies

Frequency of review

- Every 3-6 months

Clinical assessment

- blood pressure
- weight

Laboratory assessment

- urine ACR (see page 18)
- eGFR (see page 15)
- biochemical profile including urea, creatinine and electrolytes
- HbA1c (for people with diabetes)
- fasting lipids
- full blood count
- calcium and phosphate
- parathyroid hormone (6-12 monthly if eGFR < 45 mL/min/1.73m²)

Other assessments

- assess absolute cardiovascular risk (see page 8)
- blood pressure reduction (see page 10)
- lifestyle modification (see page 10)
- lipid lowering treatment (where appropriate for risk factor reduction) (see page 41)
- assess risk of atherosclerotic events and consider treating with an anti-platelet agent unless there is an increased bleeding risk
- glycaemic control (see page 37)
- avoid nephrotoxic medication or volume depletion and adjust doses to levels appropriate for kidney function (see page 21)
- assess for common complications (see pages 35-44)
- appropriate referral to Nephrologist when indicated (see page 20)
- multidisciplinary care (see page 31)

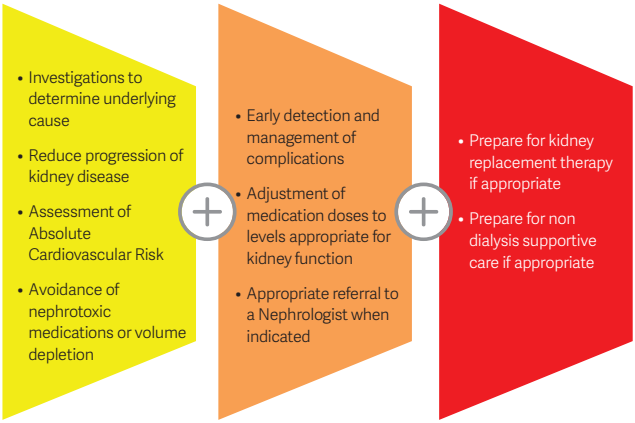
Care Plan Template available to download at www.kcat.org.au

Red clinical action plan

Macroalbuminuria irrespective of eGFR or

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

Goals of management



Management strategies

Frequency of review
- Every 1-3 months

Clinical assessment
- blood pressure
- weight
- oedema

Laboratory assessment
- urine ACR (see page 18)
- eGFR (see page 15)
- biochemical profile including urea, creatinine and electrolytes
- HbA1c (for people with diabetes)
- fasting lipids
- full blood count (if anaemic, page 35)
- calcium and phosphate
- parathyroid hormone (6-12 monthly if eGFR < 45 mL/min/1.73m²)

Other assessments
- assess absolute cardiovascular risk (see page 8)
- blood pressure reduction (see page 10)
- lifestyle modification (see page 10)
- lipid lowering treatment (where appropriate for risk factor reduction) (see page 41)
- assess risk of atherosclerotic events and consider treating with an anti-platelet agent unless there is an increased bleeding risk
- glycaemic control (see page 37)
- avoid nephrotoxic medication or volume depletion and adjust doses to levels appropriate for kidney function (see page 21)
- assess for common complications (see pages 35-44)
- appropriate referral to Nephrologist when indicated (see page 20)
- multidisciplinary care (see page 31)
- discuss treatment options, including dialysis, transplant and non dialysis supportive care if eGFR < 30 and progressing to kidney replacement therapy (see pages 24-35)
- discuss advance care plans if appropriate (see page 26)
- In patients with stage 4-5 CKD who are suitable for dialysis, the arm veins suitable for placement of vascular access should be preserved. In particular the cephalic veins of the non-dominant arm should not be used for venepuncture for blood testing or for the insertion IV catheters.

Care Plan Template available to download at www.kcat.org.au

CKD and its complications

Early detection and intervention has 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 renal acid excretion compounded by a reduction in bicarbonate production. Acidosis contributes to demineralization of bone and increased protein degradation, which may be associated with increased morbidity.

Management

- Supplementation with sodium bicarbonate (SodiBic 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 keep the HCO_3 level above 22mmol/L
  - Higher doses can be prescribed, but carry a higher risk of fluid overload
- Increased sodium load may worsen blood pressure control

Albuminuria[8]

Target:
50% reduction in urine ACR

Albuminuria is an important prognostic feature in CKD. The degree of albuminuria relates to the severity of the kidney disease and with a greater likelihood of progression to end stages of CKD. The amount of albuminuria can be reduced significantly by the use of an ACE inhibitor or ARB agent. Reduction in the amount of albuminuria is associated with improved outcomes.

Management

- ACE inhibitor or ARB as first-line therapy
- Reduction in salt output through reducing oral salt intake
- Spironolactone (use with caution on specialist advice and ensure regular monitoring of serum potassium)

Consumer fact sheet 'Albuminuria' available to download at www.kidney.org.au

Anaemia[32]

Target:
Hb 100 – 115 g/L

Prior to commencement of ESA a trial of iron supplementation maintaining: Ferritin >100 µg/L; TSAT >20%

Once ESA commenced, maintain: Ferritin 200-500 µg/L; TSAT 20-30%

- Anaemia of CKD is related to:
  - reduced erythropoietin production by the kidney
  - resistance to the action of 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 markedly with decreasing GFR.

Management

- Other forms of anaemia should be considered and excluded.
  - B12 and folate levels should be checked and corrected if deficient.

- Iron deficiency is a common cause of anaemia in people with CKD.
- If iron deficiency is identified, other cause should be excluded (e.g., blood loss).
- Prior to commencement of ESA a trial of IV iron should be considered to maintain ferritin >100 µg/L; TSAT >20%.

- 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.
- Treatment with ESA must be commenced by or in consultation with 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 pre-dialysis or peritoneal dialysis patients.
- ESAs are available either through hospital pharmacies or on Authority prescription under section 100 of the PBS for 'treatment of anaemia requiring transfusion, defined as a haemoglobin level of less than 100 g/L, where intrinsic renal disease as assessed by a Nephrologist, is the primary cause of the anaemia'. A private hospital provider number is required to access the drug on Authority prescription through a community pharmacy.
- It is recommended that ESA therapy is used with great caution, if at all, in CKD patients with active malignancy. If used in this setting, target Hb levels are lower in those patients, and the lowest dose of ESA is used to prevent blood transfusion.
- ESA treatment can be divided into two phases:
  - Correction: treatment commenced with the aim of achieving target Hb. It is reasonable in this phase to monitor Hb ~2-4 weekly and iron stores monthly. The aim is a rise of Hb at a rate of approximately 10g/L/month. Rapid correction of anaemia has been associated with hypertension and seizures.
  - Maintenance: target Hb is not fully defined in CKD, but the range is between 100-115 g/L. There is evidence of potential harm when Hb is targeted to exceed 130 g/L. Monitoring of Hb and iron studies is generally at three monthly intervals during this phase.

Consumer fact sheet 'Anaemia' available to download at www.kidney.org.au

Depression[27]

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 recurrently and maintain a high level of clinical awareness for depression.
- 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 nonmedication therapies (e.g., education, cognitive behavioural therapy, exercise programs) and antidepressant medication.
- SSRIs (selective serotonin reuptake inhibitors) have established safety in people with CKD (for a detailed list of the most common classes of antidepressant medications with suggested dosing in kidney impairment, and potential adverse effects see www.nature.com/ki/journal/v81/n3/fig_tab/ki2011358t2.html27).

Consumer fact sheet 'Depression and chronic kidney disease' available to download at www.kidney.org.au

Dietary protein[8]

Target:

No lower than 0.75 g/kg body weight/day

Dietary protein restriction has been shown to result in modest slowing of CKD progression. However, the beneficial effect of protein restriction is typically outweighed by the deleterious effects of nutritional restriction. See page 23 for more information on nutrition and CKD.

Management

- Dietary advice (refer to an Accredited Practising Dietitian)

Glycaemic control[33]

Target:

BGL: 6-8mmol/L fasting; 8-10 mmol/L postprandial

HbA1c: Generally: ≤53 mmol/mol (range 48-58); ≤7% (range 6.5-7.5). Needs individualisation according to patient circumstances (e.g., disease duration, life expectancy, important comorbidities, and established vascular complications).

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 (see General Practice Management of Type 2 Diabetes - 2014-15[33] for individualised recommendations).

Some medications may need to be reduced in dose or ceased in CKD (see page 21). See also Appendix 1 from the "Australian Diabetes Society Position Statement on A New Blood Glucose Management Algorithm for Type 2 Diabetes"[26] for a list of medication options for people with diabetes and CKD www.mja.com.au/sites/default/files/issues/201_11/gun01187_Appendix1.pdf.

Management

- Lifestyle modification (see page 10)
- Oral hypoglycaemics
- Gliptins
- Incretin mimetics
- Insulin

Consumer fact sheet 'Diabetic kidney disease' available to download at www.kidney.org.au

Haematuria[34]

- The most common causes of haematuria are non-glomerular conditions such as menstrual contamination or urological conditions (urinary tract infection (UTI), renal calculi, prostatic disease, or urinary tumours).
- Visible (or macroscopic) haematuria must always be investigated.
- Haematuria due to 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 a mild underlying glomerulonephritis with a low propensity for progression.

Management

- Use dipsticks rather than urine microscopy as dipsticks are more sensitive and accurate.
- Evaluate further if there is a result of 1+ or more.
- Do not use urine microscopy to confirm a positive result. However, urine microscopy may be useful in distinguishing glomerular haematuria from other causes.
- Persistent invisible (microscopic) haematuria in the absence of albuminuria can be differentiated from transient haematuria if 2 out of 3 reagent strip tests are positive.
- Persistent invisible haematuria, with or without albuminuria, should prompt investigation for urinary tract malignancy in appropriate age groups.
- Persistent invisible 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. Family members should also be screened for haematuria.

Consumer fact sheet 'Blood in the urine' available to download at www.kidney.org.au

Hyperkalaemia[8]

Target:
K+ ≤ 6.0 mmol/L

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

Management

- Low K+ diet (discuss with an Accredited Practising Dietitian)
- Correct metabolic acidosis (target serum HCO_3 > 22 mmol/L)
- Potassium wasting diuretics (e.g., thiazides)
- Avoid salt substitutes which may be high in K+
- Resonium A powder
- Cease ACE inhibitor/ARB/ spironolactone if K+ persistently > 6.0 mmol/L and not responsive to above therapies
- Refer to nearest Emergency Department if K+ > 6.5 mmol/L

Hypertension[13, 35]

Target:

≤ 140/90 mmHg

or ≤ 130/80 mmHg in people with albuminuria (urine ACR >3.5 mg/mmol in females and >2.5 mg/mmol in males) or diabetes

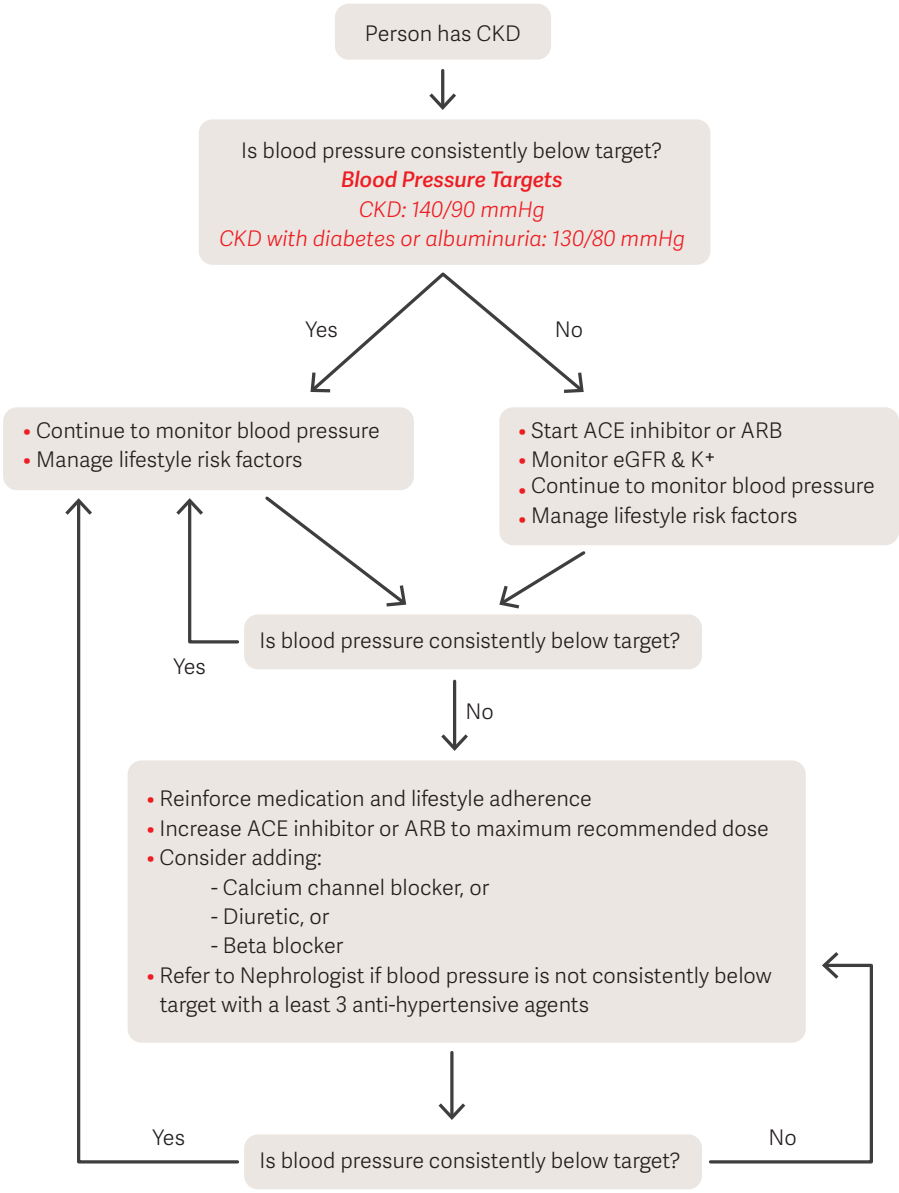
Hypertension is both a cause of CKD and a complication of CKD and can be difficult to control. The risks of uncontrolled hypertension include progression of kidney disease and increased risk of coronary heart disease and stroke. Hypertension should be considered as part of absolute cardiovascular risk (see page 8).

Management

- Lifestyle – See page 10 for guidance on basic lifestyle advice. For more detailed advice refer to relevant guidelines.
- 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.
- People with diabetes or proteinuria should be treated with an ACE inhibitor or ARB as first line therapy.
- When treatment with an ACE inhibitor or ARB is initiated, the GFR can decrease and potassium levels can rise (see page 22 for more information).
- If the serum potassium concentration is greater than 6 mmol/L despite dose reduction, diuretic therapy and dietary potassium restriction, then any ACE inhibitor, ARB or spironolactone should be stopped.
- Diuretics should be used in most individuals. Both non loop diuretics (e.g., thiazides) and loop diuretics (e.g., frusemide) are effective at all stages of CKD as adjunct antihypertensive therapy.
- Additional antihypertensive agents can be chosen based on cardiovascular indications and comorbidities.
- Beta-blockers may be useful in people with coronary heart disease, tachyarrhythmias and heart failure, but are contraindicated in asthma and heart block.
- Calcium channel blockers may be used for people with angina, the elderly and those with systolic hypertension.
- Combined therapy with ACE inhibitor and ARB is not recommended.

Consumer fact sheet 'Blood pressure and chronic kidney disease' available to download at www.kidney.org.au

Algorithm for management of hypertension in people with CKD



Lipids[36, 37]

CKD is associated commonly with substantial abnormalities of lipid metabolism, including increased low-density lipoproteins, triglycerides, very-low-density lipoproteins, and lipoprotein (a), and reduced levels of high-density lipoprotein cholesterol. Dyslipidaemia is more severe in individuals with albuminuria, particularly those with nephrotic syndrome.

Management

- 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.
- If aged ≥50 years with any stage of CKD (irrespective of lipid levels):
  - Statin if eGFR is > 60 mL/min/1.73m^2
  - Statin or statin/ezetimibe combination if eGFR is ≤ 60 mL/min/1.73m^2.
- If aged < 50 years with any stage of CKD (irrespective of lipid levels):
  - Statin if presence of one or more of: coronary disease, previous ischaemic stroke, diabetes or estimated 10-year incidence of fatal or non-fatal myocardial infarction above 10%
- Lifestyle advice if hypertriglyceridaemia is present.

Malnutrition[8, 38]

Target:

Serum albumin ≥35 g/L

Poor food intake due to the symptoms of CKD can lead to malnutrition and low serum albumin. See page 23 for more information on nutrition and CKD.

Management

- Dietary advice (refer to an Accredited Practising Dietitian)

Mineral and bone disorder[8, 39, 40]

Target:

Keep PO_4 in normal range (0.8-1.5 mmol/L)

Keep Ca in normal range (2.2-2.6 mmol/L)

Vitamin D (25-hydroxyvitamin D) levels are adequate if > 50 nmol/L

Refer to Nephrologist if PTH is persistently elevated above the upper limit of normal and rising

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 renal clearance of phosphate 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 production, and in turn elevated levels of PTH increase the resorption and release of mineral from bone. These changes are associated with an increased risk of fracture and also increased cardiovascular mortality, perhaps mediated by accelerated vascular calcification.

Management

- Phosphate
  - Dietary restriction of phosphate (refer to an Accredited Practising Dietitian).
  - Use of phosphate binders, which bind dietary phosphate to prevent absorption. Commonly used binders are typically calcium-based.
  - Sevelamer and lanthanum are available for individuals on dialysis.
- Calcium
  - If phosphate is controlled, calcium will typically remain in normal range. If the level is low with normal phosphate level consider Vitamin D supplementation.
  - Excess calcium administration should be avoided as this may be associated with increased risk of vascular calcification in CKD.
- Vitamin D
  - Cholecalciferol, the form of vitamin D that comes from sun exposure, can be given as a dietary supplement and will be converted to 25-hydroxyvitamin D by the liver.
  - If kidney function is still intact, it will then be converted to calcitriol, the most active form and will help to suppress the development of secondary hyperparathyroidism.
  - Calcitriol, the most active form of vitamin D is used in CKD for suppression of secondary hyperparathyroidism and is the preferred vitamin D in later stages of CKD when kidney function is very poor. Cholecalciferol should still be used for 25-hydroxyvitamin D deficiency in advanced CKD, including in combination with calcitriol.
  - Calcitriol is available on PBS Authority for "the indication of hypocalcaemia due to renal disease". The major side effect of therapy with calcitriol is hypercalcaemia and hyperphosphataemia.
- Cinacalcet
  - Cinacalcet, a calcimimetic agent, can be used to treat hyperparathyroidism for individuals on dialysis.
  - In people with CKD and severe hyperparathyroidism who fail to respond to medical/pharmacological therapy, parathyroidectomy should be considered, particularly when calcium or phosphate levels cannot be satisfactorily controlled.

What to measure	GFR 45-59 mL/min/1.73m^2	GFR < 45 mL/ min/1.73m^2
Calcium & phosphate	6-12 months	3-6 months
PTH & alkaline phosphatase*	Baseline	6-12 months
25-hydroxyvitamin D	Baseline	Baseline

*ALP or bone-specific ALP will help to give information on the rate of bone turnover

Consumer fact sheet 'Calcium and phosphate' available to download at www.kidney.org.au

Muscle cramps

Many people with kidney failure may experience muscle cramps due to imbalances in fluid and electrolytes, peripheral neuropathy or peripheral vascular disease.

Management

- Encourage stretching and massaging of the affected area
- Tonic water can be effective for frequent cramps

Pruritus[41]

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., allergies, scabies, inadequate dialysis, calcium/phosphate)
- 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

Restless legs

Restless Legs Syndrome (RLS) is common in CKD. As many as 8 in 10 people with eGFR < 15 mL/min/1.73m^2 have RLS or a related movement disorder called periodic limb movements in sleep (PLMS).

Management

- Check iron status and replace if deficient
- Home therapies such as massage, warm baths, warm/cool compresses, relaxation techniques, exercise
- Dopaminergic agents or dopamine agonists
- Benzodiazepines

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 10 lifestyle modification)
- Avoid central nervous system depressants (including alcohol)
- CPAP therapy (if obstructive pattern)

Uraemia

Uraemia is a syndrome seen in Stage 4 or 5 CKD, and is caused by the accumulation of the breakdown products of protein metabolism. The symptoms include anorexia, nausea, vomiting, lethargy, confusion, muscle twitching, convulsions and coma. Although urea and creatinine are the substances we measure, the symptoms are most likely due to the accumulation of other toxic end products. These symptoms can lead to poor food intake and malnutrition. By the time uraemia becomes symptomatic, dialysis is typically indicated.

Management

- Dialysis should be commenced as soon as uraemic symptoms develop
- If non-dialysis pathway is planned:
  - a low protein diet will help control gastrointestinal symptoms
  - fluid control should be strict to avoid pulmonary oedema
  - avoid unnecessary medications
  - anti-emetics are of limited value

Resources

Kidney Health Australia

www.kidney.org.au

1800 454 363 – Free call Kidney Health Information Service Line

Kidney Health Australia is a not for profit organisation whose mission is to advance the public health agenda through awareness, detection, prevention and management of kidney disease in Australia and our region.

Programs available to assist health professionals include:

- CKD-GO! Clinical Action Plan app
- Downloadable Care Plan templates
- Downloadable referral letter templates
- eGFR calculator and resources
- Interactive workshop education programs (accredited with RACGP, ACCRM, RCNA)
- Online learning modules (www.thinkgp.com.au/kha)
- Patient resources - fact sheets, brochures, books, DVDs
- Scientific reports and publications
- Renal unit locations in Australia

Kidney Check Australia Taskforce (KCAT)

KCAT education sessions support the recommendations made in this booklet and will facilitate translating these recommendations into best practice detection and management of CKD in primary care.

If you would like to undertake some education related to the contents of this booklet, please visit www.kcat.org.au for further information.

KHA-CARI Guidelines

www.cari.org.au

Evidence-based clinical practice guidelines for the management of adult and paediatric patients with CKD.

The "Early Chronic Kidney Disease" guideline is particularly relevant for primary care health professionals.

Guidelines available to download online.

Royal Australian College of General Practitioners

www.racgp.org.au

Guidelines for preventive activities in general practice (8th edition). http://www.racgp.org.au/your-practice/guidelines/redbook/

National guide to a preventive health assessment for Aboriginal and Torres Strait Islander people (2nd edition). http://www.racgp.org.au/your-practice/guidelines/national-guide/chronic-kidney-disease-prevention-and-management/

Renal Resource Centre

www.renalresource.com

A community health service of Northern Sydney Central Coast Health which provides renal patients with information and educational material to assist them in managing the effects of renal disease on their lifestyle.

Index

Abbreviations

ACE inhibitor	Angiotensin-converting enzyme inhibitor
ACRRM	Australian College of Rural and Remote Medicine
ACN	Australian College of Nursing
ACR	Albumin:creatinine ratio
AKI	Acute kidney injury
ALP	Alkaline phosphatase
APD	Automated peritoneal dialysis
APNA	Australian Primary Health Care Nurses Association
ARB	Angiotensin II receptor blocker
BMI	Body mass index
BP	Blood pressure
BSA	Body surface area
BGL	Blood glucose level
CAPD	Continuous ambulatory peritoneal dialysis
CARI	Caring for Australasians with Renal Impairment
CKD	Chronic kidney disease
CKD-EPI	Chronic Kidney Disease Epidemiology Collaboration
CPAP	Continuous positive airway pressure
CrCl	Creatinine clearance
CRP	C-reactive protein
CVD	Cardiovascular disease
DASH	Dietary Approaches to Stop Hypertension
eGFR	Estimated glomerular filtration rate
ESA	Erythropoiesis stimulating agent
ESKD	End stage kidney disease
ESR	Erythrocyte sedimentation rate
GFR	Glomerular filtration rate
Hb	Haemoglobin
HBV	Hepatitis B virus
HCV	Hepatitis C virus
HIV	Human immunodeficiency virus
HR	Hazard ratio
IV	Intravenous
KCAT	Kidney Check Australia Taskforce
KDIGO	Kidney Disease Improving Global Outcomes
KHA	Kidney Health Australia
NHMRC	National Health and Medical Research Council
NSAIDs	Non-steroidal anti-inflammatory drugs
PBS	Pharmaceutical benefits scheme
PCR	Protein:creatinine ratio
PD	Peritoneal dialysis
PKD	Polycystic kidney disease
PLMS	Periodic limb movement in sleep
PTH	Parathyroid hormone
RACGP	Royal Australian College of General Practitioners
RLS	Restless legs syndrome
Spiral CT	Spiral computed tomography
SSRI	Selective serotonin reuptake inhibitor
TSAT	Transferrin saturation
UTI	Urinary tract infection
UVB	Ultraviolet light B

Acknowledgements

Kidney Health Australia and the Kidney Check Australia Taskforce acknowledge with thanks those who contributed to the development of this resource. The Editorial Advisory Committee contained members from the existing Kidney Check Australia Taskforce committee, a multidisciplinary group with representation from kidney specialists, nurses, educators, government and consumers.

Editorial Advisory Committee:

Professor David Johnson (Chair)
Dr Kate Fawcett
Ms Barbara Harvie
A/Prof Meg Jardine
Dr Ivor Katz
A/Professor Robyn Langham
Dr Marie Ludlow (Kidney Health Australia)
A/Professor Tim Mathew (Kidney Health Australia)
Dr Craig Nelson
Dr Richard Phoon
A/Professor Kevan Polkinghorne
Dr John Saunders
Professor Tim Usherwood
Dr Sheena Wilmott

Special thanks also to A/Professor Nigel Toussaint and Professor Vlado Perkovic who provided input on the management of mineral and bone disorder and hypertension sections respectively.

AMGEN® Kidney Health Australia and the Kidney Check Australia Taskforce would like to acknowledge our sponsor, Amgen Australia, who provided an unrestricted educational grant to support the printing of this booklet.

Acknowledgements

The Chronic Kidney Disease (CKD) Management in General Practice handbook has been officially endorsed as an Accepted Clinical Resource by the Royal Australian College of General Practitioners, and has also been endorsed by the Australian Primary Health Care Nurses Association and the Australian and New Zealand Society of Nephrologists.

Citation: Chronic Kidney Disease (CKD) Management in General Practice (3rd edition). Kidney Health Australia, Melbourne, 2015.

An electronic version of this booklet is available at www.kcat.org.au

ISBN: 978-0-9808596-8-3 (print)
978-0-9808596-9-0 (web)

Reference List

(1) Australian Institute of Health and Welfare. End-stage kidney disease in Australia: total incidence, 2003-2007. Cat. no. PHE 143. Canberra: AIHW.; 2011.

(2) Kidney Disease: Improving Global Outcomes (KDIGO) Lipid Working Group. KDIGO clinical practice guideline for lipid management in chronic kidney disease. Kidney Int Suppl 2013;3:259-305.

(3) Australian Bureau of Statistics. Australian Health Survey: Biomedical Results for Chronic Diseases, 2011-12. ABS, Canberra; 2013.

(4) Australian Bureau of Statistics. Australian health survey: First Results 2011-12. 2012. Report No.: 4364.0.55.001.

(5) Australian Bureau of Statistics. Causes of Death, 2012. 2014.

(6) Tonelli M, Muntner P, Lloyd A et al. Risk of coronary events in people with chronic kidney disease compared with those with diabetes: a population-level cohort study. Lancet 2012;380(9844):807-14.

(7) Johnson DW. Evidence-based guide to slowing the progression of early renal insufficiency. Intern Med J 2004 January;34(1-2):50-7.

(8) Johnson DW, Atai E, Chan M et al. KHA-CARI Guideline: Early chronic kidney disease: Detection, prevention and management. Nephrology 2013;18:340-50.

(9) ANZDATA. The 36th Annual ANZDATA Report. Australia and New Zealand Dialysis and Transplant Registry, South Australia, Adelaide; 2014.

(10) Levey AS, de Jong PE, Coresh J et al. The definition, classification and prognosis of chronic kidney disease: a KDIGO controversies conference report. Kidney Int 2011;80(1):17-28.

(11) Anavekar NS, McMurray JJV, Velazquez EJ et al. Relation between renal dysfunction and cardiovascular outcomes after myocardial infarction. N Engl J Med 2004;351(13):1285-95.

(12) Keith DS, Nichols GA, Gullion CM, Brown JB, Smith DH. Longitudinal follow-up and outcomes among a population with chronic kidney disease in a large managed care organization. Arch Intern Med 2004 March 22;164(6):659-63.

(13) National Vascular Disease Prevention Alliance. Guidelines for the Management of Absolute Cardiovascular Disease Risk. 2012.

(14) National Vascular Disease Prevention Alliance. Guidelines for the Assessment of Absolute Cardiovascular Disease Risk. 2009.

(15) KDIGO. KDIGO 2012 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. Kidney Int Suppl 2013;3.

(16) Johnson DW, Jones GRD, Mathew TH et al. Chronic kidney disease and measurement of albuminuria or proteinuria: a position statement. Med J Aust 2012;197(4):224-5.

(17) Johnson DW, Jones GRD, Mathew TH et al. Chronic kidney disease and automatic reporting of estimated glomerular filtration rate: New developments and revised recommendations. Med J Aust 2012;197(4):222-3.

(18) Royal Australian College of General Practitioners. Guidelines for preventive activities in general practice (8th edition). The Royal Australian College of General Practitioners, South Melbourne, Australia; 2012.

(19) NACCHO/RACGP. National guide to a preventive health assessment for Aboriginal and Torres Strait Islander people (2nd edition). RACGP, South Melbourne; 2012.

(20) Australian Bureau of Statistics. Australian Aboriginal and Torres Strait Islander Health Survey: Biomedical Results, 2012-13. 2014. Report No.: 4727.0.55.003, Canberra.

(21) Creatinine Consensus Working Group. Chronic kidney disease and automatic reporting of estimated glomerular filtration rate: a position statement. Med J Aust 2005;183(3):138-41.

(22) Maple-Brown LJ, Ekinci EI, Hughes JT et al. Performance of formulas for estimating glomerular filtration rate in Indigenous Australians with and without Type 2 diabetes: the eGFR Study. Diabetic Medicine 2014;31(7):829-38.

(23) Stevens LA, Claybon MA, Schmid CA et al. Evaluation of the Chronic Kidney Disease Epidemiology Collaboration equation for estimating the glomerular filtration rate in multiple ethnicities. Kidney Int 2011;79:555-62.

(24) Witte EC, Lambers Heerspink HJ, de Zeeuw D, Bakker SJL, de Jong PE, Gansevoort RT. First morning voids are more reliable than spot urine samples to assess microalbuminuria. J Am Soc Nephrol 2009;20:436-43.

(25) National Institute for Health and Care Excellence. Chronic kidney disease: early identification and management of chronic kidney disease in adults in primary and secondary care. 2014. Report No.: NICE clinical guideline 182.

(26) Gunton JE, Cheung NW, Davis TME, Zoungas S, Colagiuri S. A new blood glucose management algorithm for type 2 diabetes: a position statement of the Australian Diabetes Society. MJA 2014;201(11):650-3.

(27) Hedayati SS, Yalamanchili V, Finkelstein FO. A practical approach to the treatment of depression in patients with chronic kidney disease and end-stage renal disease. Kidney Int 2012;81:247-55.

(28) Hoffman TC, Legare F, Simmons MB et al. Shared decision making: what do clinicians need to know and why should they bother? Med J Aust 2014;201(1):35-9.

(29) Langham RG, Bellomo R, D'Intini V et al. KHA-CARI guideline: KHA-CARI adaptation of the KDIGO clinical practice guideline for acute kidney injury. Nephrology 2014;19(5):261-5.

(30) KDIGO clinical practice guideline for acute kidney injury. Kidney Int Suppl 2012;2:1-138.

(31) Curhan GC, Aronson MD, Preminger GM. Diagnosis and acute management of suspected nephrolithiasis in adults. UpToDate 2014;Sep 2014.

(32) Kidney Disease: Improving Global Outcomes (KDIGO) Anemia Working Group. KDIGO clinical practice guideline for anemia in chronic kidney disease. Kidney Int Suppl 2012;2(4):279-335.

(33) General practice management of type 2 diabetes - 2014-15. The Royal Australian College of General Practitioners and Diabetes Australia, Melbourne; 2014.

(34) National Collaborating Centre for Chronic Conditions. Chronic kidney disease: National clinical guideline for early identification and management in adults in primary and secondary care. London, UK: Royal College of Physicians; 2008.

(35) KDIGO. KDIGO clinical practice guideline for the management of blood pressure in chronic kidney disease. Kidney Int Suppl 2012;2(5).

(36) KDIGO. KDIGO Clinical Practice Guideline for Lipid Management in Chronic Kidney Disease. Kidney Int Suppl 2013;3(3).

(37) Palmer SC, Strippoli GFM, Craig JC. KHA-CARI commentary on the KDIGO Clinical Practice Guideline for Lipid Management in Chronic Kidney Disease. Nephrology 2014;19:663-6.

(38) Ash S, Campbell K, MacLaughlin H et al. Evidence based guidelines for the nutritional management of chronic kidney disease. Nutrition and Dietetics 2006;63 (Suppl 2):S35-S45.

(39) Moe S, Drueke T, Cunningham J, Goodman W, Martin K, Olgaard K. Definition, evaluation, and classification of renal osteodystrophy: A position statement from Kidney Disease: Improving Global Outcomes (KDIGO). Kidney Int 2006;69:1945-53.

(40) National Kidney Foundation. K/DOQI clinical practice guidelines for bone metabolism and disease in chronic kidney disease. Am J Kidney Dis 2003;42:S1-S201.

(41) Brown MA, Crail SM. ANZSN renal supportive care guidelines. Nephrology 2013;18:401-54.

(42) Frisoli TM, Schmeider RE, Grodzicki T, Messerli FH. Beyond salt: lifestyle modifications and blood pressure. European Heart Journal 2011;32:3081-7.

(43) National Health and Medical Research Council. Australian Dietary Guidelines. Canberra: National Health and Medical Research Council.; 2013.

(44) National Health and Medical Research Council. Australian Guidelines to Reduce Health Risks from Drinking Alcohol. Canberra: National Health and Medical Research Council.; 2009.

Treatment targets for people with CKD

Parameter	Target	Approximate reduction in systolic BP[42]
Smoking	Stop smoking using counselling and, if required, nicotine replacement therapy or other medication.	
Nutrition	Consume a varied diet rich in vegetables, fruits, wholegrain cereals, lean meat, poultry, fish, eggs, nuts and seeds, legumes and beans, and low-fat dairy products. Limit salt to < 6 g salt per day (≤100 mmol/day). Limit foods containing saturated and trans fats. See Australian Dietary Guidelines[43].	Sodium restriction: 4-7 mHg (for reduction by 6g salt intake daily) DASH diet: 5.5 mmHg for normotensives; 11.4 mmHg for hypertensives
Alcohol	Limit alcohol intake to ≤2 standard drinks per day. See Australian Guidelines to Reduce Health Risks from Drinking Alcohol[44].	3 mmHg (for 67% reduction from baseline of 3-6 drinks per day)
Physical activity	At least 30 minutes moderate physical activity on most or preferably every day of the week.	5 mmHg
Obesity	Limit energy intake to maintain a healthy weight. Ideal weight should be BMI < 25 kg/m^2 and waist circumference < 94 cm in men (< 90 cm in Asian men) or < 80 cm in women (including Asian women).	4.4 mmHg (for 5.1kg weight lost)

The NHMRC recommends immunisation against influenza and invasive pneumococcal disease for people with diabetes and/or ESKD.

Clinical tip

People with CKD should be treated with blood-pressure lowering drugs to maintain a blood pressure that is consistently below 140/90 mmHg. If albuminuria is present (urine ACR >3.5 mg/mmol in females and >2.5 mg/mmol in males) a consistent blood pressure below 130/80 mmHg should be achieved. If diabetes is present, the blood pressure should be consistently maintained below 130/80 mmHg. Consistent blood pressure control will often require the use of more than one agent. As eGFR declines more drugs will typically be required to achieve consistent blood pressure control.

Connect with us
Freecall 1800 454 363
www.kidney.org.au

Stages of CKD

		Albuminuria Stage		
Kidney Function Stage	GFR (mL/min/1.73m²)	Normal (urine ACR mg/mmol)Male: < 2.5 Female: < 3.5	Microalbuminuria (urine ACR mg/mmol) Male: 2.5-25 Female: 3.5-35	Macroalbuminuria (urine ACR mg/mmol) Male: > 25 Female: > 35
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			

Goals of management

- Investigations to determine underlying cause
- Reduce progression of kidney disease
- Assessment of Absolute Cardiovascular Risk
- Avoidance of nephrotoxic medications or volume depletion

+

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

+

- Prepare for kidney replacement therapy if appropriate
- Prepare for non dialysis supportive care if appropriate

The Australian Kidney Foundation
Trading as Kidney Health Australia
ABN 37 008 464 426 | Charity No. CH 0614