Chronic Kidney Disease (CKD) Management in Primary Care

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

4th Edition 2020
The Chronic Kidney Disease (CKD) Management in Primary Care (4th edition) handbook has been officially recognised as an Accepted Clinical Resource by The Royal Australian College of General Practitioners (RACGP), and endorsed by the Australian Primary Health Care Nurses Association (APNA), and the Australian and New Zealand Society of Nephrologists (ANZSN).

Supporting the work of Kidney Health Australia

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A digital version of this handbook is available at www.kidney.org.au

Please refer to the digital version of the handbook for any relevant updates.
Why use this handbook?

*Chronic Kidney Disease (CKD) Management in Primary Care (4th edition)* provides best practice recommendations for detecting and managing CKD in primary care:

- Easy to use and interactive.
- Quick reference guide containing all the essential information on detecting and managing CKD.
- Colour coded CKD staging table.
- Colour coded clinical action plans outlining goals of management, key management tasks and frequency of assessment required by CKD stage.
- Medication advice and treatment targets.
- Management framework for common CKD complications.
- Nephrology referral guidelines.
- Links to fact sheets, websites, and additional resources.

The *CKD Management in Primary Care handbook* is available in both hard copy and electronic soft copy (free download from www.kidney.org.au). It is also available in app format via the app CKD Go! (free download on your app store).

What’s new in the 4th edition?

- New treatment targets for the management of hypertension in people with CKD (maintain blood pressure below 130/80mmHg for anyone with CKD).
- New quick reference guide.
- Kidney disease hotspots – are you in one?
- New algorithms, flow charts and action charts.
- New sick day plan for management of acute kidney injury.
- New information on managing CKD in the presence of diabetes and cardiovascular disease.
- New section on the management of kidney cysts.
- New information on the management of oedema and cognitive decline in CKD.
- Expanded information on ‘whole of practice approach’ to CKD care.
- Expanded information on self-management and behavioural change in CKD.
- Integrated resources and handouts.
Foreword

Welcome to the 4th edition of the Chronic Kidney Disease (CKD) Management in Primary Care handbook. With this new edition, comes an updated title that reflects the important contribution of all health professionals engaged in primary care and their role in the delivery of best-practice care for patients at risk of and living with chronic kidney disease (CKD).

CKD remains an under-recognised condition in Australia, due to the asymptomatic nature of the disease and low awareness among Australians regarding risk factors for kidney disease. Further compounding this problem is the issue of limited access and utilisation of health care services amongst those most at risk of CKD. This includes the geographically isolated, socially disadvantaged as well as Aboriginal peoples and Torres Strait Islanders. Therefore, while we increasingly focus on policy and whole of society approaches, including chronic disease care frameworks, proactive health checks and lifestyle and social determinants of chronic disease, there is much that can be done in primary care at an individual level to detect and slow progression of CKD.

This 4th edition handbook emphasises the relationship between CKD, cardiovascular disease and diabetes. Given the potential for new therapeutics that may address all three conditions, it is particularly pertinent that we no longer view the patient in disease or organ ‘silos’ but recognise that addressing multi-morbidity through patient-centred cohesive care is the most clinically effective and cost-effective approach. Primary care is the central setting where patient engagement in self-management, support for shared decision making, and multidisciplinary team care can be delivered to change the course of potentially devastating illnesses such as CKD.

This handbook continues to be an extensively utilised resource in primary care. It is both practical and accessible, and is used widely out in the field, especially when making key clinical decisions, due to its easy-to-use style and hard copy and digital formats. Many health professionals also utilise the highly regarded CKD Go! digital application, which has accompanied the handbook since 2016. Positive feedback from the clinical community strengthens our commitment to this handbook, and we are confident the changes within this new edition will enhance its utility for all primary care professionals.
We thank Kidney Health Australia’s Primary Care Education Advisory Committee (PEAK) (formerly known as KCAT) for their tireless efforts to ensure this handbook contains relevant, up-to-date and evidence-based recommendations for care that are sensible, practical and reflect approaches to care that are achievable in real-life clinical practice. The substantial expertise, wisdom and insight they bring to the table is greatly valued. Special thanks must go to eminent nephrologist Prof David Johnson, who has been the chair of PEAK for the last 13 years. His experience in international and national policy for kidney care, expertise as a clinician and researcher, and excellent leadership of the activities of PEAK are highly valued by the Australian clinical community and Kidney Health Australia.

We also acknowledge the immense contribution to Nephrology of the late A/Prof Timothy Mathew, former Medical Director of Kidney Health Australia. Under his direction, many important initiatives were undertaken that tangibly changed clinical practice, not least his role in bringing about universal eGFR reporting by Australian laboratories and the genesis of this handbook. And not lastly, thank you to the exceptional Primary Care Education team at Kidney Health Australia, they are instrumental in ensuring the success of our primary care initiatives.

A/Prof Shilpa Jesudason
Clinical Director
Kidney Health Australia

Mr Chris Forbes
Chief Executive Officer
Kidney Health Australia
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Quick reference guide - CKD essentials
**Definition of CKD**

CKD is defined as:

An estimated or measured glomerular filtration rate (GFR) <60 mL/min/1.73m² 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)

**Who is at risk of CKD?**

Adult Australians are at increased risk of developing CKD if they have any of the following risk factors:

- Diabetes
- Hypertension
- Established cardiovascular disease
- Family history of kidney failure
- Obese (body mass index ≥30 kg/m²)

- Smoker
- 60+ years or older
- Aboriginal or Torres Strait Islander origin
- History of acute kidney injury (AKI)
Detecting CKD

CKD should be detected by performing a Kidney Health Check every 1-2 years on individuals who have risk factors for CKD (see page 19 for more detail).

**Kidney health check**

- **Blood test**
  - eGFR calculated from serum creatinine

- **Urine test**
  - Albumin/Creatinine Ratio (ACR) to check for albuminuria

- **BP check**
  - Blood pressure "maintain consistently below BP goals"
Quick reference guide - CKD essentials

Algorithm for initial detection of CKD

Kidney Health Check not recommended

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

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)

Possible acute kidney injury - discuss with Nephrologist

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

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

Minimum 2 out of 3 elevated urine ACRs present for ≥3 months

Indication not present

eGFR Urine ACR
eGFR <60 mL/min/1.73m²
≥20% reduction in eGFR
Repeat eGFR within 7 days
Repeat eGFR twice within 3 months
Stable reduced eGFR
Repeat eGFR twice within 3 months
Minimum 3 reduced eGFRs present for ≥3 months

Indication not present

Albuminuria Stage

<table>
<thead>
<tr>
<th>Kidney Function Stage</th>
<th>GFR (mL/min/1.73m²)</th>
<th>Normal (urine ACR mg/mmol)</th>
<th>Microalbuminuria (urine ACR mg/mmol)</th>
<th>Macroalbuminuria (urine ACR mg/mmol)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>≥90</td>
<td>Male: &lt;2.5</td>
<td>Male: &gt;25</td>
<td>Male: &gt;25</td>
</tr>
<tr>
<td>2</td>
<td>60-89</td>
<td>Female: &lt;3.5</td>
<td>Female: &lt;25-25</td>
<td>Female: &lt;3.5-35</td>
</tr>
<tr>
<td>3a</td>
<td>45-59</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3b</td>
<td>30-44</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>15-29</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>&lt;15 or on dialysis</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

- Not CKD unless haematuria, structural or pathological abnormalities present
- 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 on page 31 for management strategies.
## Treatment goals for people with CKD

| Parameter          | Treatment goal                                                                                                                                                                                                 | For more info                                                                                     |
|--------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| **Diet and 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 <6g /day (≤100mmol/day).  
• Limit intake of foods containing saturated and trans fats.  
• Limit intake of foods containing added sugars.  
• Drink water to satisfy thirst.  
• Avoid high calorie sweetened carbonated beverages at all costs.  
• Dietary protein no lower than 0.75 g/kg body weight / day.  
• Maintain Serum albumin ≥35 g/L. | • See page 38  
• Australian dietary guidelines  
• Accredited Practising Dietitian                                                                                                 |
| **Obesity**        | • Ideal weight should be BMI ≤25.  
• Waist circumference <94cm in men (<90cm in Asian men) or <80cm in women (including Asian women).                                                                                                  |                                                                                                   |
| **Physical activity** | • Be active on most, preferably all, days every week.  
• Accumulate 150 to 300 minutes (2 ½ to 5 hours) of moderate intensity physical activity or 75 to 150 minutes (1 ¼ to 2 ½ hours) of vigorous intensity physical activity, or an equivalent combination of both moderate and vigorous activities, each week.  
• Do muscle strengthening activities on at least 2 days each week.  
• Refer to Physical Activity and Sedentary behaviour guideline for age specific recommendations. | • Australia’s physical activity and sedentary behaviour guidelines  
• Exercise physiologist                                                                                                                      |
| **Smoking**        | • Stop smoking using counselling and, if required nicotine replacement therapy or other medication.                                                                                                          | • Quitline 13 7848                                                                                 |
| **Alcohol** | Limit intake to ≤2 standard drinks per day to reduce risk of alcohol – related disease or injury over a lifetime.  
Do not drink >4 standard drinks on any single occasion. | NHMRC Australian guidelines |
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Hypertension</strong></td>
<td>Maintain below 130/80 mmHg for all people with CKD.</td>
<td>See page 45</td>
</tr>
</tbody>
</table>
| **Glycaemic control** | Blood glucose levels (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). | See page 43  
RACGP Managing Type 2 Diabetes in General Practice handbook  
Diabetes Australia |
| **Albuminuria** | 50% reduction in urine ACR. | See page 69 |
| **Lipids** | Use statin or statin/ezetimibe combination in people ≥50 years with any stage of CKD, or in people <50 years with any stage of CKD in the presence of one or more of: coronary disease, previous ischaemic stroke, diabetes or estimated high cardiovascular risk (>15% over 5 years - cardiovascular risk can be assessed using www.cvdcheck.org.au)).  
No target serum cholesterol level recommended. | KDIGO guidelines  
KHA-CARI guidelines  
National Vascular Disease Prevention Alliance  
RACGP National guide to preventive health assessment in Aboriginal and Torres Strait Islander people |
| **Anaemia** | 100-115g/L.  
Prior to commencement of erythropoietin stimulating agent (ESA) a trial of iron supplementation maintaining: Ferritin >100 µg/L.  
Once ESA commenced, maintain: Ferritin 200-500 µg/L; TSAT 20-30%. | See page 70 |
| **Potassium** | K+ ≤6.0 mmol/L. | See page 75 |
| **Immunisation** | Influenza and invasive pneumococcal disease vaccination recommended for all people with diabetes and / or ESKD. | NHMRC guidelines |
Key clinical tips

Detecting and staging CKD

- Individuals with risk factors for CKD should undergo a Kidney Health Check every 1-2 years.
- Combine estimated glomerular filtration rate (eGFR) and urine albumin/creatinine ratio (ACR) results to determine the stage of CKD.
- CKD in itself is not a diagnosis. Attempts should be made to identify the underlying cause of CKD.
- Use the CKD-EPI equation to accurately determine eGFR.
- If eGFR is <60 mL/min/1.73m², retest within 7 days and consider:
  - clinical situations where eGFR results may be unreliable and/or misleading.
  - acute kidney damage.
- An eGFR <60 mL/min/1.73m² 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.

CKD impact on cardiovascular risk

- People with moderate or severe CKD (defined as eGFR <45 mL/min/1.73 m² or persistently having a urine ACR >25 mg/mmol (males) or >35 mg/mmol (females)) are already considered to be at the highest risk of a cardiovascular event (>15% probability in five years).
- They 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 cardiovascular disease (CVD) risk in that individual.

Managing CKD

- Management of early CKD includes steps to reduce cardiovascular disease risk.
- Recommend lifestyle changes and prescribe angiotensin-converting enzyme (ACE) Inhibitors or angiotensin receptor blockers (ARBs) to lower blood pressure and slow the progression of albuminuria.
- 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.
- Care of elderly people with CKD requires an individualised approach to address comorbidities, together with variability in functional status, life expectancy and health priorities.
Medications

• Medication adjustments are often needed in people with CKD and without them kidney function can be further compromised.
• Educate your patients to flag their kidney status with other providers and ensure your patient is aware that having CKD can affect prescribing of medications.
• Consider referral to a pharmacist for a Home Medication Review.

eGFR decline with ACE inhibitors or ARBs

• 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.

Patient safety – blood pressure and pain medications (the ‘triple whammy’)

• The combination of ACE inhibitor (or ARB), diuretic and non-steroidal anti-inflammatory (NSAID) or cyclooxygenase-2 (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.

Preventing acute kidney injury in CKD

• If patients become ill and are unable to maintain adequate fluid intake, they should be advised to withhold medications which will increase the risk of decline in kidney function or have reduced clearance and increase risk for adverse events.
• ACE inhibitors and ARBs may be temporarily discontinued during acute illness, but should be recommenced when the condition stabilises.

Please note that requirements for pharmaceutical benefits scheme (PBS) subsidy may differ from recommendations contained in this guide.
Chronic kidney disease in Australia – a snapshot
Why worry about chronic kidney disease?

Chronic kidney disease is a condition that needs greater attention in Australia, despite presenting major public health challenges and costing the health system an estimated $5.1 billion per year.

As the Chief Medical Officer for the Australian Government and as nephrologist, I have seen the difference that proactive detection and management of CKD can make in outcomes for patients. Primary care health professionals have a key role to play in achieving these outcomes and I encourage you to utilise this handbook to make proactive changes to your practice and help drive change within the health of Australia.

Professor Brendan Murphy
Commonwealth Chief Medical Officer

As a CKD patient, the change I experienced from life ‘before’ end-stage kidney disease (ESKD) to living with ESKD was sudden.

With this sudden change, for me, came an acute need for information. My first source of information was the family GP and my nephrologist, then later the nurses who treated me as I commenced the inevitable dialysis.

Peter J Williams
Kidney Health Australia National Consumer Council Member
### CKD in Australia - the facts

#### Common

- **1.7 million**

  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.²

- **CKD is twice as common as diabetes.³**

- **1 in 3** Australian adults has risk factors for CKD.⁴

#### Harmful

- **People with CKD have a 2-3 fold greater risk of cardiac death than people without the condition.⁵**

- **65**

  Around 65 Australians die every day with kidney disease, which is more than breast cancer, prostate cancer, or road traffic accidents.⁶

- **CKD is a stronger risk factor for future coronary events and all-cause mortality than diabetes.⁷**

  CKD can have significant impact on work, family, and psychosocial wellbeing.

#### Treatable

- **50%**

  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.⁸

  Early management of CKD (lifestyle changes, prescription of ACE inhibitors or ARBs, optimal glycaemic control) includes cardiovascular disease risk reduction.

#### Often overlooked

- **Fewer than 10%** of the people with CKD are aware they have this condition.⁹

- **Late referral is common.** 18% of people commence dialysis within 90 days of being referred to a renal service.¹⁰
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.

Kidney disease hotspots

The prevalence of CKD does not occur in an even geographical spread across Australia. Data analyses based on data collected in the Australian Health Survey show that across the country there are a number of ‘CKD hotspots’ - geographical areas that have significantly increased prevalence of CKD.2,11 CKD hotspots and data for your local area can be found at www.aihw.gov.au/reports/chronic-kidney-disease/geographical-variation-ckd/. These maps will provide you with local insights useful for your practice planning.

Causes of end stage kidney disease (ESKD)

**The most common causes of ESKD in Australia are:**10

- Diabetes
- Glomerulonephritis
- Hypertension
- Polycystic kidney disease (PKD)

Further information and resources

- Kidney Health Australia www.kidney.org.au
  - Kidney Helpline free-call 1800 454 363; kidney.helpline@kidney.org.au
- Australian Health Survey www.abs.gov.au/australianhealthsurvey
- ANZDATA Registry www.anzdata.org.au
Detecting CKD
Who is at risk of CKD?

Adult Australians are at increased risk of developing CKD if they have any of the following risk factors:

- Diabetes
- Hypertension
- Established cardiovascular disease
- Family history of kidney failure
- Obese (body mass index ≥30 kg/m²)
- Smoker
- 60 years or older
- Aboriginal or Torres Strait Islander origin
- History of acute kidney injury (AKI)

See page 21 for information on testing people at risk of CKD.

Hypertensive disorders in pregnancy including pre-eclampsia may increase the risk of hypertension and CKD later in life.

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 9).

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
Early detection of CKD

Benefits of early detection

• 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.

• Early detection can reduce the risk of iatrogenic AKI.

Early detection using the Kidney Health Check

• Testing for CKD to allow earlier detection and management is an important and effective strategy to reduce the increasing burden of CKD.

• Testing for CKD should not be universal, but should be targeted and performed in individuals at increased risk of developing CKD.¹³

• CKD testing should include a urine ACR for albuminuria, a blood test for serum creatinine to estimate glomerular filtration rate (GFR), and blood pressure measurement – the Kidney Health Check.¹³

Clinical tip

Individuals with risk factors for CKD should undergo a Kidney Health Check every 1-2 years.
Early detection of CKD using Kidney Health Check\textsuperscript{14, 15}

<table>
<thead>
<tr>
<th>Indications for assessment*</th>
<th>Recommended assessments</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetes</td>
<td>Urine ACR, eGFR, blood pressure.</td>
<td>Every 1-2 years\textsuperscript{†}</td>
</tr>
<tr>
<td>Hypertension</td>
<td>If urine ACR positive repeat twice over 3 months (preferably first morning void). If eGFR &lt;60mL/min/1.73m\textsuperscript{2} repeat within 7 days.</td>
<td></td>
</tr>
<tr>
<td>Established cardiovascular disease**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Family history of kidney failure</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obesity (BMI ≥30 kg/m\textsuperscript{2})</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smoker</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aboriginal or Torres Strait Islander origin aged ≥30 years\textsuperscript{‡}</td>
<td></td>
<td></td>
</tr>
<tr>
<td>History of acute kidney injury</td>
<td>See recommendations on page 53</td>
<td></td>
</tr>
</tbody>
</table>

* 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 23 for more detail regarding recommendations for testing in Aboriginal and Torres Strait Islander peoples.
Diagnosing CKD

**CKD is defined as:**

An estimated or measured glomerular filtration rate (GFR) <60 mL/min/1.73m² 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)

**There are three components to a diagnosis of CKD**

\[
\text{eGFR results to give CKD Stage (1-5)} + \text{urine ACR results normo, micro or macro albuminuria?} + \text{underlying pathology to determine cause of CKD} = \text{CKD diagnosis}
\]

**Clinical tip**

CKD in itself is not a primary diagnosis. Attempts should be made to identify the underlying cause of CKD.
Aboriginal and Torres Strait Islander peoples

- Health professionals working in primary care should be aware of the cultural, spiritual and psychological health dimensions of kidney disease for Aboriginal and Torres Strait Islander peoples.
- Consider culturally appropriate communication, including the use of Indigenous health practitioners, interpreters and family support during clinical interactions.

Data from the Australian Aboriginal and Torres Strait Islander Health Survey 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.
- Aboriginal and Torres Strait Islander peoples are twice as likely to have CKD, and four times more likely to have Stages 4-5 CKD, than non-Indigenous Australians.
- 90% of Aboriginal Torres Strait Islander peoples with CKD are not aware that they have this condition.

Recommendations for CKD detection in Aboriginal and Torres Strait Islander peoples

<table>
<thead>
<tr>
<th>Indications for assessment</th>
<th>Recommended assessments</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>People aged 18-29 years without any CKD risk factors.</td>
<td>Screen for CKD risk factors (see page 19 for list of CKD risk factors).</td>
<td>As part of annual health assessment.</td>
</tr>
<tr>
<td>All people ≥30 years and People 18-29 years with one or more CKD risk factors.</td>
<td>Urine ACR, eGFR, blood pressure. If urine ACR positive repeat twice over 3 months (preferably first morning void). If eGFR &lt;60mL/min/1.73m² repeat within 7 days.</td>
<td>Every two years (or more frequently if CVD risk is elevated).</td>
</tr>
</tbody>
</table>

For further detailed information refer to the National Guide to a Preventive Health Assessment for Aboriginal and Torres Strait Islander People (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
• CKD risk checks can be conducted annually using the Aboriginal and Torres Strait Islander health check MBS 715, which provides 5 annual visits to allied health e.g. dietitian, exercise physiologist.
• Eligible for Aboriginal and Torres Strait Islander peoples specific pharmaceutical benefits.
• May be eligible for ‘Closing the Gap’ scheme with primary healthcare nurse support through MBS 10987 and Enhanced Primary Care Allied Health support through chronic disease care planning in general practice.
Tests used to investigate CKD

Glomerular filtration rate (GFR)\textsuperscript{18}

\begin{itemize}
  \item GFR is accepted as the best overall measure of kidney function.
  \item GFR can be estimated (eGFR) from serum creatinine using prediction equations.
  \item eGFR is a more sensitive marker for CKD than serum creatinine alone.\textsuperscript{18}
  \item 50% or more of kidney function can be lost before the serum creatinine rises above the upper limit of normal.
  \item Normal serum creatinine measurements do not exclude serious loss of kidney function.
\end{itemize}

**How to assess eGFR\textsuperscript{18}**

eGFR is automatically reported (using the CKD-EPI equation) with requests for serum creatinine in individuals aged ≥18 years.

\begin{itemize}
  \item The CKD-EPI equation is the preferred method for measuring eGFR and has been shown to have greater accuracy and precision compared to other formulae.
  \item Cockcroft-Gault formula is no longer recommended when calculating eGFR for the detection of CKD.
  \item Further investigation of reduced eGFR is only required if the eGFR is <60 mL/min/1.73m\textsuperscript{2}.
\end{itemize}

**Clinical situations where eGFR results may be unreliable and/or misleading:\textsuperscript{19}**

\begin{itemize}
  \item Acute changes in kidney function (e.g. AKI).
  \item People on dialysis.
  \item 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).
  \item Exceptional dietary intake (e.g. vegetarian diet, high protein diet, creatinine supplements).
  \item Extremes of body size.
  \item Conditions of skeletal muscle, paraplegia, or amputees (may overestimate eGFR).
  \item High muscle mass (may underestimate eGFR).
  \item Children under the age of 18 years.
  \item Severe liver disease present.
  \item eGFR values above 90 mL/min/1.73m\textsuperscript{2}.
  \item Drugs interacting with creatinine excretion (e.g. fenofibrate, trimethoprim).
  \item Pregnancy (see page 26).
  \item Minor changes in eGFR could be due to physiological or laboratory variability.
\end{itemize}
**eGFR and drug dosing**

- Dose reduction of some drugs is recommended for people with reduced kidney function (See page 51).
- Manufacturers’ renal dosing recommendations for medications are often based on Cockcroft-Gault estimates of creatinine clearance (CrCl mL/min).
- 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 1.73m² 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, and South-East Asian, African, Indian and Chinese individuals living in Western countries.

**eGFR and pregnancy**

- 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.
**Urine Albumin / Creatinine Ratio (ACR)**

- 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.
- 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).\(^1\)
- 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 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.

**How to detect albuminuria:**\(^13\)

- 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).\(^2\)
- 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.
Factors other than CKD known to increase urine albumin excretion:\textsuperscript{13}

- Urinary tract infection
- High dietary protein intake
- Congestive cardiac failure
- Acute febrile illness

- Heavy exercise within 24 hours
- Menstruation
- Genital discharge or infection
- Drugs (especially NSAIDs)

\textbf{Confirming CKD}

Following confirmation of decreased eGFR and/or increased urine ACR over a three month period, an individual is able to be diagnosed with CKD and their stage of CKD determined using the algorithm for initial detection of CKD.

Refer to algorithm on page 9.

* Remember to code the CKD diagnosis in your practice software!

\textbf{The following diagnostic evaluation tests for CKD are always indicated:}\textsuperscript{12}

- Renal ultrasound scan.
- Repeat (within 1 week) serum urea/electrolytes/creatinine/eGFR/albumin tests. If eGFR continues to decrease refer to AKI management plan (see page 53).
- 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:\(^\text{12}\)

<table>
<thead>
<tr>
<th>If the following is present:</th>
<th>Carry out the following test:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Signs of systemic disease (e.g. rash, arthritis, features of connective tissue disease, pulmonary symptoms or deteriorating kidney function).</td>
<td>• Anti-glomerular basement membrane antibody.</td>
</tr>
<tr>
<td>Risk factors for HBV, HCV or HIV (these conditions are associated with an increased risk of glomerular disease).</td>
<td>• Anti-neutrophil cytoplasmic antibody.</td>
</tr>
<tr>
<td>Age &gt;40 years and possible myeloma is suspected.</td>
<td>• Anti-nuclear antibodies.</td>
</tr>
<tr>
<td></td>
<td>• Extractable nuclear antigens.</td>
</tr>
<tr>
<td></td>
<td>• Complement studies.</td>
</tr>
<tr>
<td></td>
<td>HBV, HCV, HIV serology.</td>
</tr>
<tr>
<td></td>
<td>Serum and urine protein electrophoresis.</td>
</tr>
</tbody>
</table>

Further information and resources

  - Kidney Helpline free-call 1800 454 363; [kidney.helpline@kidney.org.au](mailto:kidney.helpline@kidney.org.au)
4

Managing CKD in Primary Care
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
• Smoking

Laboratory assessment
• Urine ACR (see page 27)
• eGFR (see page 25)
• Biochemical profile including urea, creatinine and electrolytes
• HbA1c (for people with diabetes)
• Fasting lipids

Other assessments
• Assess absolute cardiovascular risk (see page 41 for criteria on who to assess including age groups)
• Blood pressure reduction (see page 45)
• Lifestyle modification (see page 36)
• Lipid lowering treatment (where appropriate for risk factor reduction) (see page 75)
• Glycaemic control (see page 43)
• Avoid nephrotoxic medication or volume depletion (see page 51)
• Whole of practice approach to CKD (see page 34)
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

- 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.

Management strategies

Frequency of review
- Every 3-6 months

Clinical assessment
- Blood pressure
- Weight
- Smoking

Laboratory assessment
- Urine ACR (see page 27)
- eGFR (see page 25)
- 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 41 for criteria on who to assess including age groups)
- Blood pressure reduction (see page 45)
- Lifestyle modification (see page 36)
- Lipid lowering treatment (where appropriate for risk factor reduction) (see page 75)
- Assess risk of atherosclerotic events and consider treating with an anti-platelet agent in keeping with existing cardiovascular guidelines
- Glycaemic control (see page 43)
- Avoid nephrotoxic medication or volume depletion and adjust doses to levels appropriate for kidney function (see page 51)
- Assess for common issues (see pages 68-79)
- Appropriate referral to nephrologist when indicated (see page 61)
- Whole of practice approach to CKD (see page 34)
Red clinical action plan

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

**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.

**Management strategies**

**Frequency of review**
- Every 1-3 months

**Clinical assessment**
- Blood pressure
- Weight
- Smoking
- Oedema

**Laboratory assessment**
- Urine ACR (see page 27)
- eGFR (see page 25)
- Biochemical profile including urea, creatinine and electrolytes
- HbA1c (for people with diabetes)
- Fasting lipids
- Full blood count (if anaemic, see page 70)
- Calcium and phosphate
- Parathyroid hormone (6-12 monthly if eGFR <45 mL/min/1.73m²)

**Other assessments**
- Assess absolute cardiovascular risk (see page 41 for criteria on who to assess including age groups)
- Blood pressure reduction (see page 45)
- Lifestyle modification (see page 36)
- Lipid lowering treatment (where appropriate for risk factor reduction) (see page 75)
- Assess risk of atherosclerotic events and consider treating with an anti-platelet agent in keeping with existing cardiovascular guidelines¹
- Glycaemic control (see page 43)
- Avoid nephrotoxic medication or volume depletion and adjust doses to levels appropriate for kidney function (see page 51)
- Assess for common issues (see pages 68-79)
- Appropriate referral to nephrologist when indicated (see page 61)
- Whole of practice approach to CKD (see page 34)
- Discuss treatment options, including dialysis, transplant and non-dialysis supportive care if eGFR <30 and progressing to kidney replacement therapy
- Discuss advance care plans if appropriate (see page 66)
Whole-of-practice approach to CKD management

The management of CKD is always a collaborative effort, and a whole of practice approach involving the general practitioner (GP), primary health care nurse and practice staff maximises the opportunity for best practice care to occur. Identification of a clinical lead, clinical governance, correct coding of CKD and implementation of e-health will all impact outcomes.

As kidney function declines, and as complications and comorbidities increase, a whole-of-practice approach is even more essential for optimal care.

The role of the GP

GPs play 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 GP 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.
• co-development of a management plan with the patient that encourages and supports adherence to the lifestyle and medication plan22 (see information on MBS item numbers).
• 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.

The role of the primary health care nurse

Primary health care nurses work collaboratively with the GP in providing best practice care for people with CKD and are pivotal in:

• identifying people at risk of CKD.
• supporting patients with CKD with self-management strategies.
• facilitating individualised patient centred care planning.
• motivational interviewing to help change behaviours in a supportive way.
• providing ongoing education and support to the person with CKD.

MBS item numbers

In Australia, a number of Medicare items are designed to support proactive, integrated and multidisciplinary care for people with chronic disease.

• The use of Chronic Disease Management (CDM) item numbers such as those for GP Management Plans (GPMP) and Team Care Arrangements (TCAs) (MBS Items 721, 723, 729, 731 and 732) provide rebates to manage chronic or terminal medical conditions, including CKD, by preparing, coordinating, reviewing or contributing to CDM plans.
• They apply for a patient who suffers from at least one medical condition that has been present (or is likely to be present) for at least six months or is terminal.

• Once a GPMP and TCAs have been prepared, the patient may be eligible for access to certain individual allied health services, such as visits with a dietitian, (MBS items 10950 to 10970 inclusive) on referral from their GP.

More information can be found at www.health.gov.au/mbsprimarycareitems.

Further information and resources


• Kidney Health Australia www.kidney.org.au
  - Kidney Helpline free-call 1800 454 363; kidney.helpline@kidney.org.au

Patient-led behavioural change

Behaviour change is a complex process and empowering patients to take the lead in making behaviour changes that will benefit their health outcomes, is a useful strategy in chronic disease management.

• Health professionals should consider offering health coaching that assists the patient to self-reflect on lifestyle behaviour strategies that would work for them in managing their condition.

• It is useful to consider the ‘stages of behaviour change’ model that outlines the process individuals go through when identifying and integrating changes into their lives:
  1) pre-contemplative / unaware
  2) contemplative
  3) preparing
  4) action
  5) maintaining
  6) termination / advocacy / transcendence.

• Patients should be actively encouraged to be a partner in their care.

For further information or training consider Health Coaching courses available throughout Australia.
Lifestyle modification

Lifestyle modification should always be considered the first line management strategy for people diagnosed with CKD. Implementing lifestyle changes (Smoking, Nutrition, Alcohol, Physical activity (SNAP)) can have a positive effects on CKD outcomes and can delay the progression of the disease.

The five A’s provide a key framework for addressing lifestyle issues.

- **ask**
- **assess**
- **advise/agree**
- **assist**
- **arrange**

### Obesity
- Being obese (BMI ≥30) doubles your risk of developing CKD compared to someone of a healthy body weight.
- Central obesity is more important than generalised obesity, waist circumference is useful.
- Obese people may be more likely to develop albuminuria.

### Smoking
- Smokers have an increased risk of 40% to 105% (depending on their pack-year history) compared to non-smokers.
- Smoking has been associated with kidney damage.
- Among individuals with diabetes, those who smoke are more likely to get albuminuria and among those with diabetic nephropathy, smoking accelerates progression to failure.

### Physical activity
- Physical activity has important benefits to overall physical and emotional health and wellbeing.
- Beyond the general health effects, there is growing evidence that physical activity is beneficial in the management of many chronic conditions.
- Higher cardiorespiratory fitness levels, increased participation in physical activity and less time spent in sedentary pursuits are all associated with better CKD outcomes.
- Gradual increase in physical activity from a sedentary lifestyle is likely to be safer than remaining sedentary for most people, but risks should be assessed on an individual basis.
## Lifestyle targets for people with CKD

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Target</th>
<th>Approximate reduction in systolic BP&lt;sup&gt;29&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Smoking</strong></td>
<td>• Stop smoking using counselling and, if required, nicotine replacement therapy or other medication.</td>
<td></td>
</tr>
</tbody>
</table>
| **Nutrition**              | • Consume a varied diet rich in vegetables, fruits, wholegrain cereals, lean meats, poultry, fish, eggs, nuts and seeds, legumes and beans, and low-fat dairy products (e.g. Dietary Approaches to Stop Hypertension (DASH) diet).  
  • Limit salt to <6 g salt per day (≤100 mmol/day).                                   
  • Limit intake of foods containing saturated and trans-fats.                                                                                           
  • Limit intake of foods containing added sugars.                                                                                                        
  • See Australian Dietary Guidelines.<sup>30</sup>                                                                                                          
  • See specific information on Nutrition in CKD on page 38.                                                                                               | Sodium restriction: 4-7 mmHg (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.                                                                                   | 3 mmHg (for 67% reduction from baseline of 3-6 drinks per day).                                                                 |
  • See Australian Guidelines to Reduce Health Risks from Drinking Alcohol.<sup>31</sup>                                                                               |
| **Physical activity**      | • Be active on most, preferably all, days every week.                                                                                   | 3 - 5 mmHg.                                                                                                     |
  • Accumulate 150 to 300 minutes (2 ½ to 5 hours) of moderate intensity physical activity or 75 to 150 minutes (1 ¼ to 2 ½ hours) of vigorous intensity physical activity, or an equivalent combination of both moderate and vigorous activities, each week.  
  • Do muscle strengthening activities on at least 2 days each week.                                                                                     
  • Refer to Physical Activity and Sedentary behaviour guideline for age specific recommendations.<sup>32</sup> |                                                                                                                                 |
### Managing CKD in Primary Care

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Target</th>
<th>Approximate reduction in systolic BP&lt;sup&gt;29&lt;/sup&gt;</th>
</tr>
</thead>
</table>
| **Obesity** | • Limit energy intake to maintain a healthy weight.  
• Ideal weight should be BMI <25 kg/m<sup>2</sup>.  
• Waist circumference <94 cm in men (<90 cm in Asian men) or <80 cm in women (including Asian women). | 4.4 mmHg (for 5.1 kg weight lost). |

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

### Nutrition<sup>12</sup>

- 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 NHMRC.
- Australian guidelines recommend that people with eGFR <30 mL/min/1.73m<sup>2</sup> should have individualised diet intervention involving an Accredited Practising Dietitian.
- Overweight or obese people with CKD should be encouraged to lose weight under the management of an Accredited Practising Dietitian.

**Nutrition recommendations for people with CKD and eGFR ≥30mL/min/1.73m<sup>2</sup>.**<sup>12*</sup>

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Target</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Protein</strong></td>
<td>• Recommended daily intake: 0.75-1.0 g/kg/day.</td>
</tr>
</tbody>
</table>
| **Sodium** | • No greater than 100 mmol/day (2.3 g sodium or 6 g salt per day).  
• Avoid adding salt during cooking or at the table.  
• Choose salt reduced packaged food products.  
• Avoid salt substitutes that contain high amounts of potassium salts. |
| **Potassium** | • If persistent hyperkalaemia is present, refer to an Accredited Practising Dietitian for nutrition assessment and advice regarding dietary potassium restriction (see page 75 for further information on managing hyperkalaemia). |
| **Fluid** | • Drink water to satisfy thirst.  
• Increased fluid intake is not necessary.  
• Avoid high-calorie sweetened carbonated beverages at all costs. |

* People with eGFR <30 mL/min/1.73m<sup>2</sup> should have nutrition assessment by an Accredited Practising Dietitian.*
Further information and resources

- Kidney Health Australia  
  www.kidney.org.au
  - Nutrition resources, recipe books & videos  
  - CKD Go! App – all the best bits of this handbook in a free app - available on your app store
  - My Kidneys, My Health – Free handbook and app for people newly diagnosed with CKD  
  - Kidney Helpline free-call 1800 454 363 kidney.helpline@kidney.org.au
  - Accredited online education on detecting and managing CKD in primary care  

- RACGP Red Book  

- George Institute salt swap  
  www.georgeinstitute.org.au

- Renal Diet Diary App

- Dietitian’s Association of Australia  
  https://daa.asn.au

- NHMRC Nutrient Reference values  
  www.nrv.gov.au

- Australian Dietary Guidelines  

- Australian Guidelines to Reduce Health Risks from Drinking Alcohol  
  https://www.nhmrc.gov.au

- Australian Physical Activity guidelines  
  www.health.gov.au

- Baker Institute Lifestyle modification factsheets for chronic conditions  
  baker.edu.au/health-hub/fact-sheets

- Quitline  
  www.quit.org.au
Managing CKD in conjunction with other chronic conditions

CKD rarely occurs in isolation. In a primary care setting, it is very likely that individuals will have a CKD diagnosis that sits alongside one or more other chronic conditions.

CKD shares many treatment goals and management strategies with other common chronic conditions such as diabetes and cardiovascular disease. Taking a ‘whole of person’ approach and managing chronic conditions in conjunction with one another will lead to improved patient outcomes.

Prevalence of cardiovascular disease (CVD), diabetes, CKD, and their comorbidity*3

*For persons aged 18 and over, 2011-2012.

CKD and cardiovascular disease

- Both reduced eGFR and significant albuminuria are independent risk factors for cardiovascular disease (CVD).³³
- CKD is a more important risk factor for CVD than diabetes.⁷
- Even early stage CKD constitutes a significant risk factor for cardiovascular events and death.³⁴
- 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.³⁵

**Absolute cardiovascular risk assessment¹**

- An absolute cardiovascular risk approach evaluates the probability that an individual will develop cardiovascular disease within a given period of time.
- The assessment looks at a combination of risk factors and their intensity rather than evaluating single risk factors (such as cholesterol levels) independently.
- Using an absolute risk approach can help health professionals by providing meaningful and individualised levels of risk.
- Clinical decisions based on absolute cardiovascular risk can lead to improved health outcomes and can be useful to educate and motivate patients.
Assessing absolute cardiovascular risk

Cardiovascular risk can be assessed using the Australian Absolute Cardiovascular Risk Tool found at www.cvdcheck.org.au.

<table>
<thead>
<tr>
<th>Who SHOULD have their cardiovascular risk assessed?</th>
<th>Who SHOULD NOT have their absolute cardiovascular risk assessed?</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Adults aged ≥45 years.</td>
<td>Adults already known to be at increased absolute risk of cardiovascular disease:</td>
</tr>
<tr>
<td>• Aboriginal and Torres Strait Islander peoples aged ≥35 years.</td>
<td>• Moderate or severe CKD</td>
</tr>
<tr>
<td>Without existing cardiovascular disease.</td>
<td>- Macroalbuminuria (persistent urine ACR &gt;25 mg/mmol in males or &gt;35 mg/mmol in females) or</td>
</tr>
<tr>
<td>Not already known to be at increased risk of CVD.</td>
<td>- eGFR &lt;45 mL/min/1.73m².</td>
</tr>
<tr>
<td></td>
<td>• Diabetes and age &gt;60 years.</td>
</tr>
<tr>
<td></td>
<td>• Diabetes with microalbuminuria (persistent urine ACR &gt;2.5 mg/mmol in males or &gt;3.5 mg/mmol in females).</td>
</tr>
<tr>
<td></td>
<td>• Previous diagnosis of familial hypercholesterolaemia.</td>
</tr>
<tr>
<td></td>
<td>• Systolic blood pressure ≥180 mmHg or diastolic blood pressure ≥110 mmHg.</td>
</tr>
<tr>
<td></td>
<td>• Serum total cholesterol &gt;7.5 mmol/L.</td>
</tr>
</tbody>
</table>

Clinical tip

People with moderate or severe CKD (persistent urine ACR >25 mg/mmol (males) or >35 mg/mmol (females) or eGFR <45 mL/min/1.73m².) 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.
CKD and diabetes

- One in every two people who visits their primary care practice with type 2 diabetes will have CKD.\(^\text{36}\)
- Diabetes is a significant risk factor for CKD with up to 40% of CKD being caused by diabetes.\(^\text{10}\)
- The presence of diabetes worsens the outcomes in all stages of CKD (cardiovascular outcomes, dialysis survival, and post-transplant survival).\(^\text{37}\)
- Kidney disease is known to be the key marker of cardiovascular risk in diabetes.\(^\text{38}\)
- Early detection and appropriate treatment measures can slow or stop the progression of chronic kidney disease in people with diabetes.

**Diabetes treatment targets:**

| 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).  
 |         | interpret with caution if haemoglobin (Hb) is changing.  

**Management**

- 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.
- Some medications may need to be reduced in dose or ceased in CKD (refer to table below).
- When considering available diabetes treatment options it is important to note that the presence of CKD effectively doubles the risk of hypoglycaemia.\(^\text{40}\) The mechanisms for this are complex and relate to clearance of endogenous and exogenous insulin.
- Hypoglycaemia becomes more frequent as eGFR declines and medications may need to be adjusted accordingly.
<table>
<thead>
<tr>
<th>Medication Class</th>
<th>CKD Dosing</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metformin</td>
<td>• Reduce dose (eGFR 30-60 mL/min/1.73m²).</td>
<td>Should temporarily stop during periods of illness.</td>
</tr>
<tr>
<td></td>
<td>• Contraindicated (eGFR &lt;30 mL/min/1.73m²).</td>
<td></td>
</tr>
<tr>
<td>SGLT2 inhibitors</td>
<td>• Efficacy decreases, thus contraindication with moderate renal impairment.</td>
<td>Recent evidence suggests significant renal and cardiovascular benefits in people with CKD and diabetes.41-44 Possible side effects of UTI and euglycaemic diabetic ketoacidosis.</td>
</tr>
<tr>
<td></td>
<td>• SGLT2 inhibitors currently available in Australia (dapagliflozin, empagliflozin and ertugliflozin) are contraindicated for eGFR persistently &lt;45 mL/min/1.73m²). Check individual product information and PBS listings for updates.</td>
<td></td>
</tr>
<tr>
<td>Glitpkins (DPP4-inhibitors)</td>
<td>• Safe with dose adjustment.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• No dose adjustment for linaglaptin.</td>
<td></td>
</tr>
<tr>
<td>Sulphonylurea (SU)</td>
<td>• Dose reduction required at eGFR &lt;30 mL/min/1.73m².</td>
<td>Hypoglycaemia risk increases as eGFR declines. Avoid glibenclamide if eGFR &lt;60 mL/min/1.73m².</td>
</tr>
<tr>
<td>GLP-1 receptor agonist</td>
<td>• Contraindication (eGFR &lt;30 mL/min/1.73m²).</td>
<td>Potential cardiovascular benefits.</td>
</tr>
<tr>
<td>Insulin</td>
<td>• Normal doses titrated to blood sugar level.</td>
<td>As eGFR declines, risk of hypoglycaemia increases.</td>
</tr>
</tbody>
</table>
CKD and hypertension\textsuperscript{1, 45}

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 41).

**Hypertension treatment targets**

<table>
<thead>
<tr>
<th>Who?</th>
<th>Target</th>
</tr>
</thead>
<tbody>
<tr>
<td>All people with CKD</td>
<td>Maintain blood pressure consistently below 130/80 mmHg\textsuperscript{*}</td>
</tr>
</tbody>
</table>

\textsuperscript{*} Some recent evidence and clinical guidelines suggest aiming for a lower blood pressure target (systolic BP <120 mmHg) in people with CKD who are at high CVD risk may improve outcomes.\textsuperscript{46-49}

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

Lower blood pressure targets needs to be balanced with an increased risk of side effects including hypotension leading to increased falls, syncope, electrolyte abnormalities and increased episodes of AKI.
Algorithm for management of hypertension in people with CKD

Person has CKD

Is blood pressure consistently below target?

- Blood pressure target
  - <130/80 mmHg

Yes

- Continue to monitor blood pressure
- Manage lifestyle risk factors

No

- Start ACE inhibitor or ARB
- Monitor eGFR & K+
- Continue to monitor blood pressure (consider home monitoring)
- Manage lifestyle risk factors

Is blood pressure consistently below target?

Yes

- Reinforce medication and lifestyle adherence
- Increase ACE inhibitor or ARB to maximum recommended dose
- Consider adding:
  - Calcium channel blocker, or
  - Diuretic, or
  - Beta blocker
- Refer to Nephrologist if blood pressure is not consistently below target with at least 3 anti-hypertensive agents

No

Is blood pressure consistently below target?

Yes

No
Management

- Reducing blood pressure to below target levels is one of the most important goals in management of CKD.
- Lifestyle changes should always be advocated and can have a significant effect on blood pressure (see the table on page 37 for guidance on basic lifestyle advice).
- 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.
- When treatment with an ACE inhibitor or ARB is initiated, the GFR can decrease and potassium levels can rise (see page 48 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.
- Multiple medications (often 3 or more drugs) are needed to control hypertension adequately in most people with CKD.
- Additional antihypertensive agents can be chosen based on cardiovascular indications and comorbidities.
- Assess risk of atherosclerotic events and consider treating with an anti-platelet agent unless there is an increased bleeding risk.50
- Consider sleep apnoea as a cause of resistant hypertension.

Blood Pressure Monitoring

- 24 hour ambulatory blood pressure monitoring (ABPM) or home blood pressure monitoring (HBPM) have been shown to better correlate with target organ damage and cardiovascular mortality and morbidity when compared to office BP measurements.
- Use of ABPM or HBPM can also aid in the diagnosis of masked hypertension, over treatment (hypotension) and monitor response to antihypertensive treatment.
- HBPM when combined with education may increase adherence and improve overall blood pressure control.
- Where feasible HBPM should be considered to aid in the diagnosis and management of hypertension.
Commonly used anti-hypertensive medications

<table>
<thead>
<tr>
<th>Medication</th>
<th>Notes on use</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>ACE inhibitors or ARBs</strong></td>
<td>Essential part of the best care approach for many patients in all stages of CKD.</td>
</tr>
<tr>
<td></td>
<td>• They cause a reduction in glomerular blood flow, and GFR can decline when treatment is initiated.</td>
</tr>
<tr>
<td></td>
<td>• Providing the reduction is less than 25% within two months of starting therapy, the ACE inhibitor or ARB should be continued.</td>
</tr>
<tr>
<td></td>
<td>• 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.</td>
</tr>
<tr>
<td></td>
<td>• Combined therapy with ACE inhibitor and ARB should be avoided except with specialist advice.</td>
</tr>
<tr>
<td></td>
<td>• Caution should be exercised if baseline potassium is $\geq 5.5$ mmol/L, as rises in serum potassium of approximately 0.5 mmol/L are expected (see page 75).</td>
</tr>
<tr>
<td></td>
<td>• ACE inhibitors and ARBs can be safely prescribed at all stages of CKD and should not be deliberately avoided just because GFR is reduced.</td>
</tr>
<tr>
<td><strong>Non-loop diuretics</strong></td>
<td>Effective in all stages of CKD as adjunct antihypertensive therapy, particularly if fluid overload is clinically present.</td>
</tr>
<tr>
<td>(e.g. thiazides)</td>
<td></td>
</tr>
<tr>
<td><strong>Loop diuretics</strong></td>
<td></td>
</tr>
<tr>
<td>(e.g. frusemide)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• In general, it is reasonable use non-loop diuretics if the eGFR is $&gt;45$ mL/min/1.73m² and loop diuretics if the eGFR is $&lt;45$ mL/min/1.73m².</td>
</tr>
<tr>
<td></td>
<td>However:</td>
</tr>
<tr>
<td></td>
<td>• Frusemide can be used safely for management of fluid overload in all stages of CKD, including when GFR is severely reduced to $&lt;30$ mL/min/1.73m².</td>
</tr>
<tr>
<td></td>
<td>• Typical doses are 20-120 mg/day, but higher doses (up to 500 mg/day) may be required, especially at lower levels of eGFR.</td>
</tr>
<tr>
<td></td>
<td>• When more than 80 mg/d is required, the efficacy is improved by dividing the daily dose.</td>
</tr>
<tr>
<td></td>
<td>• Thiazides can be effective at low levels of eGFR, particularly in combination with loop diuretics.</td>
</tr>
<tr>
<td></td>
<td>For more information on managing oedema, see page 77.</td>
</tr>
<tr>
<td>Medication</td>
<td>Notes on use</td>
</tr>
<tr>
<td>----------------------------------</td>
<td>--------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Beta-blockers</td>
<td>Atenolol and metoprolol are useful agents for blood pressure control in people with CKD, but are contraindicated in asthma and heart block. Atenolol is renally cleared. For use of beta blockers in heart failure, refer to the National Heart Foundation Australia guidelines.51</td>
</tr>
<tr>
<td>Calcium channel blockers</td>
<td>May be used for people with angina, the elderly and those with systolic hypertension.</td>
</tr>
<tr>
<td>Mineralocorticoid receptor blockers</td>
<td>May be used for people with heart failure or hypertension. Caution in CKD and in combination with RAS blockade due to risk of decline in eGFR and hyperkalaemia. Monitor biochemistry carefully.</td>
</tr>
</tbody>
</table>

**Clinical tip**

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

**Clinical tip**

ACE inhibitors and ARBs cause a reversible reduction in glomerular blood flow and GFR can decline when treatment is initiated. 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.
Further information and resources

- Australian Absolute Cardiovascular Risk tool; resources for health professionals and patients - www.cvdcheck.org.au
- Kidney Health Australia www.kidney.org.au
  - CKD Go! App – all the best bits of this handbook in a free app (available on your app store)
  - Kidney Helpline free-call 1800 454 363; kidney.helpline@kidney.org.au
- National Heart Foundation www.heartfoundation.org.au
- Diabetes Australia www.diabetesaustralia.com.au
- Stroke Foundation www.strokefoundation.org.au
- Royal Australian College of General Practitioners (RACGP)
- Australian Diabetes Society www.diabetessociety.com.au
  Various Position Statements and a useful list of medication options for people with diabetes and CKD
- St Georges Renal - home blood pressure monitoring stgrenal.org.au/forms
Medication considerations in CKD

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/1.73m².
- Home Medicines Reviews and Residential Medication Management Reviews support general practitioner/pharmacist collaboration and are funded by Medicare item numbers.
- Remember to code CKD correctly. Your practice software can help with medication considerations!
- Educate your patients to flag their kidney status with other providers and ensure your patient is aware that having CKD can affect prescribing of medications. Refer to AKI sick day management plan on page 53 for further information.

<table>
<thead>
<tr>
<th>Commonly prescribed drugs that may need to be reduced in dose or ceased in CKD include, but are not limited to:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acarbose</td>
</tr>
<tr>
<td>Apixaban</td>
</tr>
<tr>
<td>Dabigatran</td>
</tr>
<tr>
<td>Exenatide</td>
</tr>
<tr>
<td>Gabapentin</td>
</tr>
<tr>
<td>Glimepiride</td>
</tr>
<tr>
<td>Lithium</td>
</tr>
<tr>
<td>Opioid analgesics</td>
</tr>
<tr>
<td>Saxagliptin</td>
</tr>
<tr>
<td>Spironolactone</td>
</tr>
<tr>
<td>Vildagliptin</td>
</tr>
</tbody>
</table>

* Metformin should be used with caution if GFR 30-60 mL/min/1.73m², and is not recommended if GFR <30 mL/min/1.73m². It should be temporarily interrupted during periods of ill health and/or change in kidney function.

* Trimethoprim can raise creatinine but has no effect on GFR.
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**
Patient safety: 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 patient is volume-depleted or CKD is present. Ensure individuals on ACE inhibitor or ARB plus diuretic blood pressure medication are aware of the need to discuss appropriate pain relief medication with a general practitioner or pharmacist.

**Further information and resources**

  - Australian resource focusing on drug therapy in people with CKD. Virtual tours and copies available to order online.
- ‘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](http://www.nature.com/ki/journal/v81/n3/fig_tab/ki2011358t2.html).
**Acute Kidney Injury (AKI)**\(^{54, 55}\)

- AKI is a common syndrome, especially in hospitalised patients, and is independently and strongly associated with increased morbidity and mortality.
- 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.
- AKI is diagnosed either by detection of a sudden increase in serum creatinine, OR with persistent oliguria (see below).
- Primary care practices are in a unique position to identify people at increased risk of AKI and address potentially modifiable exposures to prevent the occurrence of AKI.

### Risk factors for AKI

<table>
<thead>
<tr>
<th>Pre-existing risk factors</th>
<th>Modifiable kidney insults</th>
</tr>
</thead>
<tbody>
<tr>
<td>CKD</td>
<td>Hypovolaemia</td>
</tr>
<tr>
<td>Other chronic disease</td>
<td>- sepsis</td>
</tr>
<tr>
<td>- diabetes</td>
<td>- critical illness</td>
</tr>
<tr>
<td>- heart/lung/liver disease</td>
<td>- circulatory shock</td>
</tr>
<tr>
<td>- cancer</td>
<td>- burns</td>
</tr>
<tr>
<td>- anaemia</td>
<td>- trauma</td>
</tr>
<tr>
<td>Advanced age</td>
<td>Drugs (e.g., triple whammy)</td>
</tr>
<tr>
<td>Female gender</td>
<td>Radiocontrast agents</td>
</tr>
<tr>
<td></td>
<td>Poisonous animals (e.g., snakes, spiders)</td>
</tr>
<tr>
<td></td>
<td>Heatwave</td>
</tr>
</tbody>
</table>
## AKI prevention and management plan

<table>
<thead>
<tr>
<th>Identifying those at risk</th>
<th>How to diagnose AKI</th>
<th>What to do during an AKI episode</th>
<th>What to do after an AKI episode</th>
</tr>
</thead>
<tbody>
<tr>
<td>• All people with CKD stage 3-5 are at increased risk of AKI.</td>
<td>• Increase in serum creatinine ≥26.5 µmol/l within 48 hours or</td>
<td>• Removal of risks in early stage of illness.</td>
<td>• Annual Kidney Health Check for subsequent 3 years.</td>
</tr>
<tr>
<td>• Minimize use of NSAIDs and other potentially nephrotoxic drugs in people with CKD.</td>
<td>• Increase in serum creatinine to ≥1.5 times baseline, which is known or presumed to have occurred within the prior 7 days or</td>
<td>• Seek specialist advice early.</td>
<td>• Education and self-management to monitor and reduce risk of subsequent exposures.</td>
</tr>
<tr>
<td>• Early identification of people at risk with acute illness, and consider temporary cessation of ACE Inhibitor/ARB/diuretics with hypovolaemia/hypotension.</td>
<td>• Significant reduction in urine output compared with normal output.</td>
<td>• Systematic fluid assessment and medication review for all people at risk when acute illness occurs.</td>
<td>• Record in practice records as AKI (resolved).</td>
</tr>
</tbody>
</table>

### Preventing AKI in individuals with CKD who are sick or dehydrated

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

<table>
<thead>
<tr>
<th>Increase risk of decline in kidney function</th>
<th>Have reduced clearance and increase risk for adverse effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Angiotensin-converting enzyme inhibitors</td>
<td>• Metformin</td>
</tr>
<tr>
<td>• Angiotensin receptor blockers</td>
<td>• Sulfonylureas</td>
</tr>
<tr>
<td>• Non-steroidal anti-inflammatory drugs</td>
<td></td>
</tr>
<tr>
<td>• Diuretics</td>
<td></td>
</tr>
<tr>
<td>• SGLT2 inhibitors</td>
<td></td>
</tr>
</tbody>
</table>
Mnemonic for drugs to be avoided on a sick day (SAD MANS)

- **S**ulfonylureas
- **A**CE-inhibitors
- **D**iuretics
- **M**etformin
- **A**ngiotensin receptor blockers
- **N**on-steroidal anti-inflammatory
- **S**GLT2 inhibitors

Further information and resources

  - Kidney Helpline free-call 1800 454 363; [kidney.helpline@kidney.org.au](mailto:kidney.helpline@kidney.org.au)
Kidney stones

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

Prevention of recurrence

- 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.
- 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 (depending on stone type) should be commenced if there is evidence of continued new stone formation or if there is no or little improvement in the baseline urine chemistries with fluid and diet changes:
  - thiazides to reduce calcium excretion
  - allopurinol to reduce hyperuricosuria
  - citrate for hypocitraturia.
Acute management

- The acute management of a stone episode is usually undertaken in an emergency department with urologist involvement.
- The management of a stone episode where the stone is known to be of a size able to be spontaneously passed (<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.

Further information and resources

  - Kidney Helpline free-call 1800 454 363; kidney.helpline@kidney.org.au
Kidney cysts

Simple cysts

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

Simple cysts are:

• Very common (not inherited).
• Usually asymptomatic.
• Simple cysts can occur with advancing age.
• May be associated with background CKD.
• Do not cause kidney failure.

Indications for further review and investigation:

• Multiple cysts.
• Bilateral multiple cysts.
• Cysts with complex internal structure or solid components.
• Inability to differentiate cysts from obstruction.
• Past history of malignancy.
• Symptoms from cyst (discomfort, haematuria, infection).

Polycystic kidney disease (PKD)

• Polycystic kidney disease (PKD) is a group of chronic kidney diseases with formation of multiple cysts in the kidney.
• PKD is the most common inherited kidney disease.
• PKD is a common cause of CKD.
Consider a diagnosis of PKD if:

<table>
<thead>
<tr>
<th>Age</th>
<th>Number of cysts shown on ultrasound</th>
</tr>
</thead>
<tbody>
<tr>
<td>15-39 years</td>
<td>At least 3 in total</td>
</tr>
<tr>
<td>Aged 40-59 years</td>
<td>At least 2 in each kidney</td>
</tr>
<tr>
<td>Aged 60 years or older</td>
<td>At least 4 in each kidney</td>
</tr>
</tbody>
</table>

Clinical management of autosomal dominant polycystic kidney disease (ADPKD)*

1. Assess if at high-risk for ESKD.
2. Reduce kidney cyst growth and prevent eGFR decline and hypertension.
3. Evaluate for other kidney complications.
4. Discuss other problems.

*Refer to the KHA-CARI PKD guidelines for detailed management advice

Treatments for ADPKD

The medication tolvaptan is now listed on the PBS for the treatment of adults with early stage CKD (stage 2 to 3) and rapidly progressing ADPKD. It has been shown to slow the progression of cyst development and kidney disease in ADPKD. Refer to the PBS guidelines for guidance on which patients can be prescribed tolvaptan and the relevant rules of prescribing.

Further information and resources

- Kidney Health Australia www.kidney.org.au
  - Kidney Helpline free-call 1800 454 363; kidney.helpline@kidney.org.au
- KHA-CARI Guidelines www.cari.org.au
- PKD Foundation of Australia pkdaustralia.org.au
Progressive CKD
Indications for referral to a nephrologist\textsuperscript{12, 58}

Appropriate referral is associated with positive outcomes, including:

\begin{itemize}
\item Reduced rate of progression to ESKD.
\item Decreased morbidity and mortality.
\item Decreased need for and duration of hospitalisation.
\item Increased likelihood of timely preparation of permanent dialysis access prior to dialysis onset.
\item Increased likelihood of kidney transplantation.
\end{itemize}

\textbf{Nephrology referral recommended}

- \textbf{eGFR <30 mL/min/1.73m² (Stage 4 or 5 CKD of any cause).}
- \textbf{Persistent significant albuminuria (urine ACR ≥30 mg/mmol).}
- \textbf{A sustained decrease in eGFR of 25\% or more within 12 months OR a sustained decrease in eGFR of 15 mL/min/1.73m² per year.}
- \textbf{CKD with hypertension that is hard to get to target despite at least three anti-hypertensive agents.}

\textbf{Referral not necessary*}

- \textbf{Stable eGFR ≥30 mL/min/1.73m².}
- \textbf{Urine ACR <30 mg/mmol (with no haematuria).}
- \textbf{Controlled blood pressure.}

* In the absence of other referral indicators.

* 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.
Tests recommended 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.

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.

Further information and resources
- For a sample referral letter template, visit www.kidney.org.au/health-professionals

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.
- A shared decision making approach between patients, families and health professionals is highly recommended.
- This is best supported by a decision aid, such as the My Kidneys My Choice decision aid, available at www.kidney.org.au/your-kidneys/support/kidney-disease/treatment/choosing-your-treatment.
- Renal Supportive Care (RSC) is a program that is embedded in usual renal care along all points of the treatment pathway.
- For patients receiving renal replacement therapy (dialysis, transplant) RSC is provided in addition to usual renal care, and does not replace this care.
- RSC involves an interdisciplinary approach that integrates the skills of renal medicine and palliative care to help patients with CKD and ESKD to live as well as possible. This is achieved by better managing their symptoms and supporting them in living with advanced disease.
### Brief comparison of treatment options

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Types</th>
<th>Involves</th>
<th>Lifestyle impact/outcomes</th>
</tr>
</thead>
</table>
| **Comprehensive conservative kidney care** | • No dialysis or transplant.  
• Managed in the community.  
• Supported by palliative care or renal supportive care teams. | • Medication and diet control.  
• Advance care planning.  
• Assessing symptoms and managing with non-dialysis therapies. | • In most people, life expectancy will be decreased compared with dialysis or transplant.  
• Dialysis therapy may not be associated with a survival or quality of life advantage compared with non-dialysis supportive care in elderly people with two or more comorbidities.  
• Symptom Management (without dialysis) may improve quality of life. |
| **Home peritoneal dialysis (PD)** | Continuous ambulatory peritoneal Dialysis (CAPD). | • Four or more daytime bags changed manually. | • Need PD catheter.  
• Simple, gentle and portable.  
• 1 week training.  
• Infection risk: 1 per 2 years.  
• 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. |
### Treatment Types

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Types</th>
<th>Involves</th>
<th>Lifestyle impact/ outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Home haemodialysis</strong></td>
<td>• Daytime, 3-5 treatments weekly, 4-6 hrs duration.</td>
<td>• Blood cleansed by artificial filter.</td>
<td>• Average of 3 months for training.</td>
</tr>
<tr>
<td></td>
<td>• Night-time, 3-5 nights per week, 8 hrs. duration.</td>
<td>• Surgery for fistula at least 3 months prior to use.</td>
<td>• Flexible daily routine.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• As above, with more hours of dialysis offering better health outcomes.</td>
</tr>
<tr>
<td><strong>Centre based haemodialysis</strong></td>
<td>• Hospital or satellite centre.</td>
<td>• As above.</td>
<td>• Strict routine.</td>
</tr>
<tr>
<td></td>
<td>• 3 x weekly.</td>
<td></td>
<td>• Strict diet.</td>
</tr>
<tr>
<td></td>
<td>• 4-6 hrs (individualised).</td>
<td></td>
<td>• Transport to hospital or satellite centre needed.</td>
</tr>
<tr>
<td></td>
<td>• Occasional clinics offer overnight.</td>
<td></td>
<td>• No training required.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Infection risk (poorer outcomes than home-based forms of dialysis)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(peritoneal dialysis or home haemodialysis).</td>
</tr>
<tr>
<td><strong>Transplant</strong></td>
<td>• Living donor.</td>
<td>• Surgery.</td>
<td>• Freedom to work and travel once kidney function stabilised.</td>
</tr>
<tr>
<td></td>
<td>• Deceased donor.</td>
<td>• Lifetime immunosuppressants.</td>
<td>• Need to maintain a healthy diet, but no other restrictions.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• May wait 3-7 years for a deceased donor.</td>
<td>• Survival rates good</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Compatible live donor.</td>
<td>• Higher infections and cancer rate.</td>
</tr>
</tbody>
</table>

### Further information and resources

- Kidney Health Australia
  - www.kidney.org.au
  - Introduction to Kidney Disease Treatment Options (series of books) www.kidney.org.au/about-us/resources-library


- Kidney Helpline free-call 1800 454 363; kidney.helpline@kidney.org.au


- Your local renal unit.
Shared decision making

- 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 appropriate treatments.

Benefits include:
- Acknowledges patient values and preferences.
- Enhances patient engagement.
- Improves patient knowledge.
- Reflects evidence-based care.

Although shared decision making can occur without tools, various decision support tools now exist.

Five questions that clinicians can use to guide shared decision making

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?

Further information and resources

  - Kidney Helpline free-call 1800 454 363; kidney.helpline@kidney.org.au
- Palliative Care Australia [www.palliativecare.org.au](http://www.palliativecare.org.au)
Advance care planning

- 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.

Further information and resources

- Visit www.advancecareplanning.org.au for information and resources.

Considerations in older people

- Care of elderly people with CKD requires an individualised approach to address comorbidities, together with variability in functional status, life expectancy and health priorities.
- Relying on creatinine alone causes under-recognition of CKD.
- eGFR (which is adjusted for age) improves diagnostic accuracy.

Clinical tip

An eGFR <60 mL/min/1.73m² 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.

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.kidney.org.au/your-kidneys/support/kidney-disease/treatment/choosing-your-treatment
Appropriate referral

- Elderly patients with a stable eGFR ≥30 mL/min/1.73m², 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.
- The decline in eGFR can be variable and may depend on age, acute events and other factors. Patients with eGFR <30mL/min/1.73m² should be referred to a nephrologist and have a plan created for frequency of monitoring.

Manage cardiovascular risk

- In people with CKD, death from cardiovascular disease is more common than ESKD at all ages.
- Manage cardiovascular risk (see page 41, 37) 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.

Further information and resources

- Kidney Health Australia www.kidney.org.au
  - Kidney Helpline free-call 1800 454 363; kidney.helpline@kidney.org.au
- Dementia Australia - Mini Mental State Examination to measure global cognitive function https://www.dementia.org.au/information/for-health-professionals/clinical-resources/cognitive-screening-and-assessment
Common issues in CKD
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/1.73m² 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 HCO₃ 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**

Albuminuria is an important prognostic feature in CKD. The degree of albuminuria relates to the severity of the kidney disease and greater likelihood of progression to ESKD. 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).

**Target:**

50% reduction in urine ACR.
**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/1.73m². 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, causes including GI blood loss should be considered and excluded.
  - 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 10 g/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.

For further guidance on Anaemia in CKD refer to the KHA-CARI guidelines at www.cari.org.au

**Cognitive decline**

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

The presence of CKD:

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

**Management**

- Cognition affects many aspects of CKD care.
- Things to consider in primary care assessments are:
  - Screen for cognition in CKD – Mini-Mental State Examination (MMSE).
  - Safety.
  - Medication adherence.
  - Medication review.
  - Falls.
  - Risk of delirium.
  - Association with depression.
  - Self-care issues and engagement with care.

**Depression**

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

**Management**

- Screen regularly and maintain a high level of clinical awareness for depression. Consider use of DAS-21 or Kessler K10.
- Modifiable causes of depression that are commonly experienced by people with CKD (e.g. insomnia, medication side-effects, inadequate dialysis) should be considered and excluded.
- Treatment of persistent depressive symptoms involves a combination of non-medication therapies (e.g. education, cognitive behavioural therapy, exercise programs) and antidepressant medication.
• Selective serotonin reuptake inhibitors (SSRIs) have established safety in people with CKD.53 (For a detailed list of the most common classes of antidepressant medications with suggested dosing in kidney impairment, and potential adverse effects refer to the article referenced).

• In patients with chronic condition/s, depression and anxiety is often an impairment to their self-management strategies. Consider in General Practice Mental Health Care Plan referral to Psychologist for psychological support.

Dietary protein

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. Many patients with more advanced CKD experience anorexia and may become protein malnourished. See page 38 for more information on nutrition and CKD.

Management
• Nutrition assessment by an Accredited Practising Dietitian.

Further information and resources
• Beyond Blue - Depression and Anxiety Scale www.beyondblue.org.au/the-facts/anxiety-and-depression-checklist-k10
• Black Dog institute - Self Depression test and resources www.blackdoginstitute.org.au/clinical-resources/depression/depression-self-test
Haematuria

- 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.

Assessment

- Use dipsticks for haematuria 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.

Management

- 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.
Algorithm for the management of persistent microscopic haematuria

Persistent microscopic haematuria

Rule out infection

Actions:
- Check urine microscopy and culture

Infection present:
Mange urinary tract infection and re-test

No infection present

Is there associated albuminuria and / or reduced eGFR?

Yes

Consider Glomerular causes

Actions:
- Consider underlying glomerulonephritis
- Urinary tract ultrasound
- Manage according to CKD action plan
- Referral to nephrologist

No

Consider urologic malignancy if risk factors* present

Actions:
- Urine cytology x 3
- Urinary tract ultrasound
- Referral to urologist for consideration of cystoscopy

* Risk factors: male; age >40; history of macroscopic haematuria; smoking; pelvic irritation; exposure to occupational chemicals dyes or cyclophosphamide.
Hyperkalaemia

**Target:**
Potassium ≤6.0 mmol/L.

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

Hyperkalaemia, especially potassium levels >6.5 mmol/L, predisposes to cardiac arrhythmias.

**Management**

**Potassium 6.0 – 6.5 mmol/L:**
- Low K⁺ diet (discuss with an Accredited Practising Dietitian).
- Correct metabolic acidosis (target serum HCO₃ >22 mmol/L).
- Potassium wasting diuretics (e.g. thiazides).
- Avoid salt substitutes which may be high in K⁺.
- Consider a cation exchange resin (e.g. Resonium A).
- Cease ACE inhibitor/ARB/spironolactone if K⁺ persistently >6.0 mmol/L and not responsive to above therapies.

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

Lipids

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/1.73m².
  - Statin or statin/ezetimibe combination if eGFR is ≤60 mL/min/1.73m².
- 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 high cardiovascular risk (>15% over 5 years -cardiovascular risk can be assessed using [www.cvdcheck.org.au](http://www.cvdcheck.org.au)).
- Lifestyle advice if hypertriglyceridaemia is present.
Malnutrition\textsuperscript{12, 65}

**Target:**
Serum albumin $\geq 35$ g/L.

Poor food intake due to anorexia in CKD can lead to malnutrition and low serum albumin. See page 38 for more information on nutrition and CKD.

**Management**
- Dietary advice (refer to an Accredited Practising Dietitian).

Mineral and bone disorder\textsuperscript{12, 66, 67}

- Changes in the metabolism of calcium, phosphate, parathyroid hormone and Vitamin D typically start to occur once GFR $\leq 60$ 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 reabsorption 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**
- The management of CKD Bone Mineral Disorder is complex and should usually occur via a nephrologist.
- CKD Bone Mineral Disorder does not usually need to be tested for or addressed in CKD stage 1-3.
- Bone density can be challenging to interpret in advanced CKD and typical osteoporosis therapies may not be useful.
- Denosumab for osteoporosis has a higher risk of hypocalcaemia in CKD. It is recommended that the use of Denosumab is discussed with a Nephrologist in advanced CKD and that hypocalcaemia is corrected prior to initiation of therapy and calcium monitored for up to 12 weeks.\textsuperscript{68}
- Patients should be provided counselling on hypocalcaemia symptoms (e.g. tingling, twitching, paraesthesia, and confusion).

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.
Oedema

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

Clinical Tips

- Oedema may manifest most commonly as ankle (pedal) oedema.
- Ankle oedema may occur even if the patient is centrally volume depleted – it is not always a sign of fluid overload and does not always need to be treated.
- The clinical assessment should include blood pressure, respiratory examination when assessing patients with ankle oedema.
- Hypertension is common in fluid overload.
- Pulmonary oedema may be a feature of more advanced CKD.
- Ascites may be seen in severe fluid overload.
- Biomarkers such as brain natriuretic peptide (BNP) to assess fluid shifts may be unreliable in patients with CKD.

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

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

Ankle oedema

- Mild ankle oedema that is not symptomatic does not usually need to be treated.
- May be managed conservatively with raising legs, using stockings and moderate sodium restriction.
- Diuretic therapy with loop and thiazide diuretics should be used for treating ankle oedema only after assessment of volume status has occurred.

Pulmonary oedema

- Will usually warrant diuretic therapy.
- Significant pulmonary oedema usually requires hospital care.

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

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

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

Management

- Ensure that there are no other causes for pruritus (e.g. skin disease, scabies, inadequate dialysis, calcium/phosphate abnormalities).
- 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/1.73m² 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.
- Low dose dopaminergic agents or dopamine agonists.
- Benzodiazepines.
- Pramipexole.

Sleep apnoea

Sleep apnoea can affect up to 50% of people with eGFR <15 mL/min/1.73m² and is a significant cause of refractory hypertension.

Management

- Weight reduction (see page 34 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, and in the advanced stages – confusion (encephalopathy), muscle twitching, pericarditis, fluid overload, convulsions and coma. Although urea and creatinine are the substances measured, the symptoms are most likely due to the accumulation of other unmeasured toxic end products. By the time uraemia becomes symptomatic, dialysis is typically indicated.

Management

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

Further information and resources

• Kidney Health Australia www.kidney.org.au
  - CKD Go! App – all the best bits of this handbook in a free app available on your app store
  - Kidney Helpline free-call 1800 454 363; kidney.helpline@kidney.org.au
• KHA-CARI guidelines www.cari.org.au
# Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tr>
<td>ACE inhibitor</td>
<td>Angiotensin-converting enzyme inhibitor</td>
</tr>
<tr>
<td>ACRRM</td>
<td>Australian College of Rural and Remote Medicine</td>
</tr>
<tr>
<td>ACN</td>
<td>Australian College of Nursing</td>
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<tr>
<td>ACR</td>
<td>Albumin: creatinine ratio</td>
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<tr>
<td>AKI</td>
<td>Acute kidney injury</td>
</tr>
<tr>
<td>ALP</td>
<td>Alkaline phosphatase</td>
</tr>
<tr>
<td>APD</td>
<td>Automated peritoneal dialysis</td>
</tr>
<tr>
<td>APNA</td>
<td>Australian Primary Health Care Nurses Association</td>
</tr>
<tr>
<td>ARB</td>
<td>Angiotensin II receptor blocker</td>
</tr>
<tr>
<td>BMI</td>
<td>Body mass index</td>
</tr>
<tr>
<td>BP</td>
<td>Blood pressure</td>
</tr>
<tr>
<td>BSA</td>
<td>Body surface area</td>
</tr>
<tr>
<td>BGL</td>
<td>Blood glucose level</td>
</tr>
<tr>
<td>CAPD</td>
<td>Continuous ambulatory peritoneal dialysis</td>
</tr>
<tr>
<td>CARI</td>
<td>Caring for Australasians with Renal Impairment</td>
</tr>
<tr>
<td>CDM</td>
<td>Chronic disease management</td>
</tr>
<tr>
<td>CKD</td>
<td>Chronic kidney disease</td>
</tr>
<tr>
<td>CKD-EPI</td>
<td>Chronic Kidney Disease Epidemiology Collaboration</td>
</tr>
<tr>
<td>COX-2</td>
<td>Cyclooxygenase-2 inhibitor</td>
</tr>
<tr>
<td>CPAP</td>
<td>Continuous positive airway pressure</td>
</tr>
<tr>
<td>CrCl</td>
<td>Creatinine clearance</td>
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<tr>
<td>CRP</td>
<td>C-reactive protein</td>
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<tr>
<td>CVD</td>
<td>Cardiovascular disease</td>
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<tr>
<td>DASH</td>
<td>Dietary Approaches to Stop Hypertension</td>
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<tr>
<td>DVT</td>
<td>Deep vein thrombosis</td>
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<tr>
<td>eGFR</td>
<td>Estimated glomerular filtration rate</td>
</tr>
<tr>
<td>ESA</td>
<td>Erythropoiesis stimulating agent</td>
</tr>
<tr>
<td>ESKD</td>
<td>End stage kidney disease</td>
</tr>
<tr>
<td>ESR</td>
<td>Erythrocyte sedimentation rate</td>
</tr>
<tr>
<td>GFR</td>
<td>Glomerular filtration rate</td>
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References

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Disclaimer

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

Kidney Health Australia

www.kidney.org.au

As the peak body for kidney health in Australia, we strive to create a healthier community through increased awareness and early detection of kidney disease. We connect kidney patients to vital resources and services to help them manage their condition and improve their quality of life; and, we support and foster advocacy and research to drive improvements in the diagnosis, management and eventual cure of kidney disease.

Primary care education and resources

- Free accredited face-to-face and online education for health professionals.
- Accredited Quality Improvement Activities.
- CKD Ambassador program.
- CKD Management handbook and CKD-Go! app.
- Educational videos.
- Nephrology referral guidelines and downloadable referral letter templates.
- eGFR calculator and resources.
- Scientific reports and publications.

Our support services

- Educational resources on kidney health and kidney disease, covering diagnosis, treatment options and management.
- Fact sheets, books and educational videos about kidney disease.
- A large range of self-management resources including recipe books and nutrition resources.
- Renal unit locations guide.
- Holiday dialysis bus.
- Transplant housing.
- Support groups.
- Kidney Health Week – national kidney awareness week.
- Regular newsletters.

Kidney Helpline

- 1800 454 363
- kidneyhelpline@kidney.org.au

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

**KHA-CARI guidelines**
www.cari.org.au

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

**Royal Australian College of General Practitioners**
www.racgp.org.au


**Health Pathways**

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

**Practice Software (e.g. PAT CAT)**

Practice software such as PAT CAT can be a useful practice tool for identifying people in your practice at risk of or with CKD. Correct coding of CKD combined with the utilisation of PAT CAT searches are useful in setting up quality improvement frameworks within practice.
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

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

Possible acute kidney injury - discuss with Nephrologist

- eGFR <60 mL/min/1.73m²
- ≥20% reduction in eGFR

- 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 3 reduced eGFRs present for ≥3 months

Minimum 2 out of 3 elevated urine ACRs present for ≥3 months

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 on page 31 for management strategies