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

CHRONIC KIDNEY DISEASE (CKD) MANAGEMENT IN GENERAL PRACTICE
<table>
<thead>
<tr>
<th>Topic</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is Chronic Kidney Disease (CKD)?</td>
<td>2</td>
</tr>
<tr>
<td>Early detection of CKD using Kidney Health Check</td>
<td>6</td>
</tr>
<tr>
<td>Tests used to investigate CKD</td>
<td>7</td>
</tr>
<tr>
<td>eGFR clinical action plan</td>
<td>13</td>
</tr>
<tr>
<td>Indications for referral to a nephrologist</td>
<td>14</td>
</tr>
<tr>
<td>CKD Management</td>
<td></td>
</tr>
<tr>
<td>Stage 1 and 2</td>
<td>16</td>
</tr>
<tr>
<td>Stage 3</td>
<td>18</td>
</tr>
<tr>
<td>Stage 4</td>
<td>20</td>
</tr>
<tr>
<td>Stage 5</td>
<td>22</td>
</tr>
<tr>
<td>Treatment targets for people with CKD</td>
<td>24</td>
</tr>
<tr>
<td>Common CKD complications</td>
<td>25</td>
</tr>
<tr>
<td>Multidisciplinary care</td>
<td>32</td>
</tr>
<tr>
<td>Resources</td>
<td>34</td>
</tr>
<tr>
<td>Abbreviations</td>
<td>36</td>
</tr>
</tbody>
</table>

A Summary Guide of this booklet “Chronic Kidney Disease (CKD) Management in General Practice” may be downloaded free of charge from the Health Professionals section of the Kidney Health Australia website at [www.kidney.org.au](http://www.kidney.org.au).
- CKD is a silent condition, but can be readily detected with tests for proteinuria, haematuria, and eGFR

- CKD is becoming increasingly common due to our ageing population and a rising incidence of Type 2 diabetes

- CKD is a potent independent risk factor for cardiovascular disease

- Optimal management of the risk factors for cardiovascular disease also slows the progression of CKD

- The majority of people with CKD will be managed in general practice, not by specialists
What is Chronic Kidney Disease (CKD)?

CKD is defined as:\(^1\):

**Glomerular filtration rate (GFR)**
\(< 60 \text{mL/min/1.73m}^2\) that is present for \(\geq 3\) months with or without evidence of kidney damage,

or

**Evidence of kidney damage with or without decreased GFR** that is present for \(\geq 3\) months as evidenced by any of the following:

- microalbuminuria
- proteinurias
- glomerular haematuria
- pathological abnormalities
  (e.g. abnormal renal biopsy)
- anatomical abnormalities
  (e.g. scarring seen on imaging or polycystic kidneys)

**Clinical Tip**

If the eGFR is \(\geq 60 \text{mL/min/1.73m}^2\), and there is no evidence of kidney damage, then CKD is not present.

Common causes of CKD

The most common reasons why people start dialysis or kidney transplantation in Australia are\(^2\):

- diabetic nephropathy (32% of all new patients)
- glomerulonephritis (24%)
- hypertension (14%)
- reflux nephropathy (3%)

While the causes of end stage kidney disease are well known, the causes of CKD are not established. Irrespective of the underlying cause of CKD the treatment follows the principles outlined in this booklet.
Australian population surveys have revealed that CKD is more common than you may think.

- 1 in 3 adults are at increased risk of developing CKD
- 1 in 7 adults have some sign of CKD

Symptoms of CKD may not appear until kidney function is severely and irreversibly impaired.


- Individuals with CKD have a 2 to 3-fold greater risk of cardiac death than individuals without CKD.\(^3,4\)
- Recent studies have confirmed that even early CKD constitutes a significant risk factor for cardiovascular events and death.\(^4–6\)
- For people with CKD, the risk of dying from cardiovascular events is 20 times greater than requiring dialysis or transplantation.\(^7\)

Who is at risk of CKD?

**Modifiable risk factors:**
- smoking
- diabetes
- high blood pressure
- obesity

**Non-modifiable risk factors:**
- age over 50 years
- family history of kidney disease
- Aboriginal or Torres Strait Islander heritage

Importance of early detection

- Increasing amounts of protein in the urine correlate directly with an increased rate of progression into end-stage kidney disease
- The amount of proteinuria/albuminuria in the urine can be reduced significantly by the use of an ACE inhibitor or ARB agent
- Reduction in the amount of proteinuria is associated with improved outcomes
- Early intervention can reduce CKD progression and cardiovascular risk by 50%\(^8\), and improves quality of life

Screening for CKD

- Certain factors are associated with an increased risk of developing CKD
- All people attending their general practitioner should be assessed for CKD risk factors as part of routine primary health encounters
- Evidence supports a targeted opportunistic screening program among high-risk individuals to identify those with CKD
Using eGFR to detect CKD

- Estimated Glomerular Filtration Rate (eGFR) using the MDRD formula is the recommended method of measuring kidney function
- eGFR may be markedly reduced while the serum creatinine is still in the normal range
- An eGFR is automatically provided with every laboratory request for a serum creatinine (in people aged ≥ 18 years)
- eGFR values are automatically reported up to 90 mL/min/1.73m². Values greater than this are reported as > 90 mL/min/1.73m²
- Knowledge of eGFR between 60–90mL/min/1.73m² may be of assistance in providing an earlier warning of eGFR reduction and allowing monitoring of trends over time
- Further investigation of eGFR is only required if the eGFR is < 60mL/min/1.73m²

Serum creatinine does not increase beyond normal limits until more than 50% of GFR has been lost

![Graph showing the relationship between GFR and serum creatinine](image-url)
CKD is generally asymptomatic.
- Patients do not normally present with symptoms of CKD, so annual checking of those at risk is essential
- People with CKD may not notice any symptoms until they reach end stage kidney disease requiring dialysis or transplant (eGFR < 15 mL/min/1.73m²)

Symptoms of end stage kidney disease include:
- nocturia
- malaise
- anorexia/nausea/vomiting
- pruritus
- restless legs
- dyspnoea

Early detection of CKD using kidney health check

<table>
<thead>
<tr>
<th>Who is at higher risk of kidney disease?</th>
<th>What should be done?</th>
<th>How often?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age &gt; 50 years</td>
<td>blood pressure</td>
<td>Every 12 months</td>
</tr>
<tr>
<td>Diabetes</td>
<td>urine dipstick (microalbuminuria if diabetes present)</td>
<td></td>
</tr>
<tr>
<td>High blood pressure</td>
<td>eGFR</td>
<td></td>
</tr>
<tr>
<td>Smoking</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obesity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Family history of kidney disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aboriginal or Torres Strait Islander</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Tests used to investigate CKD

**URINE TESTS: PROTEINURIA**

- Proteinuria is a key marker of kidney damage
- Increasing amounts of protein in the urine correlate directly with an increased rate of progression to end-stage kidney disease
- Microalbuminuria (albumin excretion above the normal range but below the level of detection by tests for total protein) is a sensitive indicator of CKD in people with diabetes, and indicates an increased risk of micro and macro vascular disease that requires aggressive intervention

**Clinical Tip**

**Screen annually for proteinuria among people at risk of CKD, except for those with diabetes who should be screened for microalbuminuria.**

- The amount of proteinuria/albuminuria can be reduced significantly by the use of an ACE inhibitor or ARB agent
- Reduction in the amount of proteinuria is associated with improved clinical outcomes
- Once protein in the urine has been detected, quantitative measurements are necessary to precisely determine the protein excretion for prognostic purposes

**HOW TO DETECT AND QUANTIFY PROTEINURIA**

- Urine dipsticks are quick, cheap and readily accessible
- Should be performed at least annually for any person at increased risk of CKD
- Proteinuria present if the dipstick is 1+ or more\(^9\)
- Protein/creatinine ratio on a random spot urine sample is recommended to further quantify proteinuria\(^10\)
- Where this is abnormal a repeat specimen a few weeks later is recommended to determine if it is persistent
- People with diabetes should have tests for microalbuminuria performed at least annually by albumin/creatinine ratio (using early morning spot urine sample)
- If test positive for microalbuminuria, two further samples should be sent for albumin/creatinine ratio within two months
Clinical tip

The daily protein excretion (g/24hrs) can be estimated from the protein:creatinine ratio (measured in mg/mmol).

Example:
- Urine protein = 900 mg/L, urine creatinine = 6 mmol/L
- Urine protein:creatinine ratio = 900/6 = 150 mg/mmol
- daily protein excretion ≈ 150 x 10 = 1500 mg = 1.5 g/24 hrs

DEFINITIONS OF ALBUMINURIA AND PROTEINURIA

<table>
<thead>
<tr>
<th>Albumin/creatinine ratio</th>
<th>Microalbuminuria</th>
<th>Macroalbuminuria</th>
<th>Proteinuria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Females: 3.6–35 mg/mmol</td>
<td>Females: &gt;35 mg/mmol</td>
<td></td>
<td>–</td>
</tr>
<tr>
<td>Males: 2.6–25 mg/mmol</td>
<td>Males: &gt;25 mg/mmol</td>
<td></td>
<td>–</td>
</tr>
<tr>
<td>Dipstick &gt;3 mg/dL (albumin specific dipstick)</td>
<td>&gt;20 mg/dL (albumin specific dipstick)</td>
<td>Dipstick = 1 + or more</td>
<td></td>
</tr>
<tr>
<td>Protein/creatinine ratio</td>
<td>–</td>
<td>–</td>
<td>&gt;30 mg/mmol</td>
</tr>
<tr>
<td>24 hour protein</td>
<td>–</td>
<td>–</td>
<td>&gt;0.3 g/24 hrs</td>
</tr>
</tbody>
</table>

URINE TESTS: HAEMATURIA

- In many people, haematuria is related to menstruation or urinary tract infection (UTI)
- Persistent haematuria, or haematuria found in conjunction with other indicators of kidney damage necessitates investigation
- ‘Isolated microscopic haematuria’ refers to when haematuria is the only abnormality, and there is no albuminuria, blood pressure is normal, and eGFR > 60 mL/min/1.73m²
- Glomerular haematuria is due to kidney disease
- Non-glomerular haematuria may be due to urological conditions (UTI, renal calculi, prostatic disease, urinary tract tumours) or menstrual contamination
**HOW TO DETECT HAEMATURIA**

- Urine dipsticks are very sensitive and can identify all significant bleeding
- A positive dipstick for blood should be repeated (between menstrual periods) and then confirmed with urine microscopy
- A culture should be performed to exclude infection
- Urine phase contrast microscopy can be used to differentiate between glomerular and non-glomerular haematuria (fresh specimen required)

**ALGORITHM FOR MANAGEMENT OF PERSISTENT MICROSCOPIC HAEMATURIA**

1. Persistent positive urine dipstick for blood
2. Urine microscopy for RBC morphology
3. **Glomerular bleeding**
   - 'Isolated' microscopic haematuria
     - Do not refer
     - Monitor 2 yearly
   - Glomerular bleeding with proteinuria or reduced GFR or hypertension
     - Refer to nephrologist
4. **Non-glomerular bleeding**
   - Spiral CT
     - Spiral CT negative
     - Spiral CT shows lesion
       - Refer to specialist
5. > 40 yrs of age or risk factors for bladder cancer
   - Cystoscopy
   - Urine cytology X3
     - Cytology negative
       - Follow up at intervals for 3 years
6. < 40 yrs of age and no risk factors for bladder cancer
   - Do not refer
   - Monitor 2 yearly
BLOOD TESTS: SERUM CREATININE

- Unreliable and insensitive marker for mild to moderate chronic kidney disease
- Affected by many factors other than kidney function and varies with age, gender and muscle mass
- **Patients may lose 50% or more of their kidney function before the serum creatinine value rises above the upper limit of normal**
- Serum creatinine concentration is useful for following the trend of kidney function, in an individual over time

BLOOD TESTS: ESTIMATED GLOMERULAR FILTRATION RATE (eGFR)

- GFR is widely accepted as the best measure of kidney function
- GFR can be estimated (eGFR) from serum creatinine using predictive equations
- eGFR is recommended to be automatically reported (using Modification of Diet in Renal Disease (MDRD) equation) with every request for serum creatinine in adults aged > 18 years\(^1\)\(^2\)
- MDRD is more accurate than the Cockcroft-Gault equation among elderly and obese people, and is also more accurate as kidney function declines
- eGFR values are automatically reported up to 90 mL/min/1.73m\(^2\). Values greater than this are reported as > 90 mL/min/1.73m\(^2\)
- Knowledge of eGFR between 60–90mL/min/1.73m\(^2\) may be of assistance in providing an earlier warning of eGFR reduction and allowing monitoring of trends over time
- Further investigation of eGFR is only required if the eGFR is < 60mL/min/1.73m\(^2\)
- In healthy adults the eGFR falls by up to 10 mL/min/1.73m\(^2\) per decade beyond the age of 40 – BUT reduced eGFR is associated with cardiovascular risk for all ages
- In people aged over 70 years of age, eGFR values between 45 and 59 mL/min/1.73m\(^2\) should be interpreted with caution. If other signs of kidney damage (e.g. proteinuria, haematuria etc) are not present, a stable eGFR in this range may be consistent with normal GFR for this age and an absence of CKD related complications
INTERPRETING eGFR IN SPECIAL SITUATIONS

Clinical situations where eGFR results may be unreliable and/or misleading include:

- acute changes in kidney function (e.g. acute kidney failure)
- dialysis-dependent patients
- exceptional dietary intake (e.g. vegetarian diet, high protein diet, recent consumption of cooked meat, creatine supplements)
- extremes of body size
- diseases of skeletal muscle, paraplegia, or amputees (may underestimate) or high muscle mass (may overestimate)
- children under the age of 18 years
- severe liver disease present

USE OF eGFR IN DIFFERENT ETHNIC POPULATIONS

- The original MDRD formula contains a factor to be applied to African-American subjects raising the possibility that other variations in the formula may be required for optimal performance in different racial groups
- Pending publication of validation studies it is recommended Australasian laboratories continue to automatically report eGFR in Aboriginal and Torres Strait Islander peoples and other ethnic groups

eGFR AND DRUG DOSING

- Where an eGFR (using MDRD) is on hand it is clinically appropriate to use this to assist drug dosing decision making
- For critical dose drugs, particularly in the hospital setting, it remains important to adhere to the published recommendations
- Published recommendations usually involve the use of the Cockcroft-Gault equation to estimate eGFR, or to measure creatinine clearance in order to amend dosing for renal function
FURTHER EVALUATION

If the eGFR is < 60mL/min/1.73m² further assessment is recommended:

- blood pressure
- dipstick for haematuria and proteinuria
  - random spot urine protein/creatinine ratio if dipstick positive for protein
  - random spot urine albumin/creatinine ratio for people with diabetes even if dipstick negative for protein
- confirmatory urea/electrolytes/creatinine
- fasting lipids
- fasting glucose
- full blood count

If the abnormal eGFR is confirmed on repeat testing, kidney ultrasound should be considered.

Depending on the age of the patient and the severity of CKD, consideration may also be given to iron studies, serum calcium, phosphate and parathyroid hormone.
**eGFR clinical action plan**

* imaging or biopsy abnormalities, or proteinuria/haematuria

** hypertension, diabetes, smoker, age > 50 yrs, obesity, family history of kidney disease, Aboriginal and Torres Strait Islander people

<table>
<thead>
<tr>
<th>eGFR mL/min/1.73m²</th>
<th>Description</th>
<th>Clinical Action Plan</th>
</tr>
</thead>
</table>
| 90                | Stage 1 CKD – kidney damage* with normal kidney function | Further investigation for CKD may be indicated in those at increased risk**:  
– blood pressure  
– assessment of proteinuria  
– urinalysis  
Cardiovascular risk reduction:  
– blood pressure  
– lipids  
– blood glucose  
– lifestyle modification (smoking, weight, physical activity, nutrition, alcohol) |
| 60–89             | Stage 2 CKD – kidney damage* with mild ↓ kidney function | As above, +:  
– monitor eGFR three monthly  
– avoid nephrotoxic drugs  
– prescribe antiproteinuric drugs (ACE inhibitors or ARBs) if appropriate  
– address common complications  
– ensure drug dosages appropriate for level of kidney function  
Consider indications for referral to a nephrologist |
| 30–59             | Stage 3 CKD – moderate ↓ kidney function | As above + referral to nephrologist is usually indicated for physical and psychosocial preparation for renal replacement therapy (dialysis, preemptive transplantation, transplantation) or conservative medical management |
| 15–29             | Stage 4 CKD – severe ↓ kidney function | As above + referral to nephrologist to end-stage disease |
| <15               | Stage 5 CKD – end-stage kidney disease | As above + referral to a nephrologist |
Indications for referral to a Nephrologist

Appropriate referral is associated with:
- reduced rates of progression to end stage kidney disease
- decreased need for and duration of hospitalisation
- increased likelihood of permanent dialysis access created prior to dialysis onset
- reduced initial costs of care following the commencement of dialysis
- increased likelihood of kidney transplantation
- decreased patient morbidity and mortality

WHO MAY BE CONSIDERED FOR REFERRAL TO A NEPHROLOGIST?

Anyone with:
- eGFR < 30mL/min/1.73m²
- Unexplained decline in kidney function (> 15% drop in eGFR over three months)
- Proteinuria > 1g/24hrs (see clinical tip)
- Glomerular haematuria (particularly if proteinuria present)
- CKD and hypertension that is hard to get to target
- Diabetes with eGFR < 60mL/min/1.73m²
- Unexplained anaemia (Hb < 100 g/L) with eGFR < 60mL/min/1.73m²

Anyone with an acute presentation and signs of acute nephritis should be regarded as a medical emergency and referred without delay.

Clinical tip

Urine protein:creatinine ratio of 100 mg/mmol ≈ daily protein excretion of 1g/24hrs.
WHO DOES NOT USUALLY NEED TO BE REFERRED TO A NEPHROLOGIST?

CKD Stage 2 and 3

- Stable eGFR 30–89 mL/min/1.73m²
- Minor proteinuria
  (<0.5 g/24hrs with no haematuria)
- Controlled blood pressure

The decision to refer or not must always be individualised, and particularly in younger patients the indications for referral may be less stringent (e.g. minor proteinuria).

In CKD Stages 2 and 3

- Don’t refer to nephrologist if targets of therapy are achieved
- Pay attention to CVD risk reduction
- Use ACE inhibitors or ARBs
- Monitor three to six monthly

Clinical tip

When referring to a nephrologist, ensure patient has had a recent kidney ultrasound, current blood chemistry, and quantification of proteinuria.
CKD Management: Stage 1 and 2 (eGFR ≥ 60 mL/min/1.73m²)

GOALS OF STAGE 1–2 CKD MANAGEMENT
- investigations to exclude treatable kidney disease
- reduce progression of kidney disease
- reduce cardiovascular risk

MONITORING IN STAGE 1–2 CKD
When monitoring patients with Stage 1–2 CKD consider:
- 3–6 monthly clinical review
- clinical assessment
  - blood pressure
  - weight
  - urine dipstick
- laboratory assessment
  - biochemical profile including urea, creatinine and electrolytes
  - eGFR
  - fasting glucose
  - fasting lipids

CARDIOVASCULAR RISK REDUCTION

The presence of CKD is one of the most potent known risk factors for cardiovascular disease.⁶,¹³

All people with CKD should undergo cardiovascular and kidney disease risk factor modification.¹,¹⁴

Individuals with CKD have a 10–20 fold greater risk of cardiac death than age and sex matched controls without CKD.⁴

People with CKD are at least 20 times more likely to die from cardiovascular disease than survive to need dialysis or a transplant.⁷

Lifestyle modification
- Lifestyle modification: cessation of smoking, weight reduction, low-salt diet, physical activity, and moderate alcohol consumption are successful in reducing overall cardiovascular risk
- Refer to SNAP guide for detection and management of lifestyle risk factors.¹⁵
Blood pressure reduction
- CKD can cause and aggravate hypertension, and hypertension can contribute to the progression of CKD
- Reducing blood pressure to target levels is one of the most important goals in management of CKD\textsuperscript{16}
- ACE inhibitors are recommended as first line therapy. ARBs may provide similar kidney protection\textsuperscript{17}
- Maximal tolerable doses of ACE inhibitors or ARBs are recommended
- Hypertension may be difficult to control and multiple (three to four) medications are frequently required\textsuperscript{8}

Clinical Tip
- ACE inhibitors and ARBs can cause an increase in serum creatinine when treatment is initiated
- If the increase in creatinine is less than 30% and stabilises within two months of starting therapy, medication should be continued\textsuperscript{18}
- If the rise in creatinine level exceeds 30% above the baseline value, medication should be ceased and consideration given to investigating for bilateral renal artery stenosis\textsuperscript{18}

Lipid-lowering treatments
- Statin therapy produces a modest reduction in proteinuria and results in a small reduction in the rate of kidney function loss, especially in people with cardiovascular disease\textsuperscript{19}

Glycaemic control
- For people with diabetes, intensive blood glucose control significantly reduces the risk of developing CKD\textsuperscript{20–23}, and in those with CKD reduces the rate of progression
Goals of Stage 3 CKD Management

- Reduce progression of kidney disease
- Reduce cardiovascular risk
- Early detection and management of complications
- Avoidance of renally-excreted and nephrotoxic medications
- Adjustment of medication doses to levels appropriate for kidney function
- Appropriate referral when indicated

Monitoring in Stage 3 CKD

When monitoring patients with Stage 3 CKD consider:

- One to three monthly clinical review
- Clinical assessment
  - Blood pressure
  - Weight
  - Urine dipstick
- Laboratory assessment
  - Biochemical profile including urea, creatinine and electrolytes
  - eGFR
  - Fasting glucose
  - Fasting lipids
  - Full blood count
  - Iron stores
  - Calcium and phosphate
  - Parathyroid hormone (quarterly)

Medications Review

Dosage reduction or cessation of renally excreted medications is generally required once the GFR falls below 60 mL/min/1.73m². It is important to review renally excreted medications, as well as avoid nephrotoxic medications in people with CKD.
Commonly prescribed drugs that need to be reduced in dose or ceased in people with CKD:
- Colchicine
- Digoxin
- Famciclovir
- Gabapentin
- Glibenclamide
- Glimepiride
- Lithium
- Metformin (significantly increased risk of lactic acidosis when GFR < 50 mL/min/1.73m²)
- Sotalol
- Valaciclovir

Commonly prescribed drugs that can adversely affect kidney function in people with CKD:
- NSAIDs and COX–2 inhibitors
- Beware the ‘triple whammy’ of NSAID/COX–2 inhibitor, ACE inhibitor and diuretic (low dose aspirin is okay)
- Radiographic contrast agents
- Aminoglycosides
- Lithium

Clinical Tip
- The combination of ACE inhibitor (or ARB), diuretic and NSAID (except low-dose aspirin) can result in a potentially fatal interaction, the ‘triple whammy’
- Ensure your patients on blood pressure medication are aware of the need to discuss pain relief medication with a general practitioner or pharmacist

EFFECT OF ACE INHIBITORS AND ARBS ON KIDNEY FUNCTION
- When treatment with an ACE inhibitor or ARB is initiated, the creatinine levels can rise.
- Refer Clinical Tip in CKD Management Stage 1 and 2
GOALS OF STAGE 4 CKD MANAGEMENT

– referral to a nephrologist for physical and psychosocial preparation for renal replacement therapy (dialysis, pre-emptive transplantation, transplantation) or conservative management
– reduce progression of kidney disease
– reduce cardiovascular risk
– early detection and management of complications
– avoidance of renally-excreted and nephrotoxic medications
– adjustment of medication doses to levels appropriate for kidney function

MONITORING IN STAGE 4 CKD

When monitoring patients with Stage 4 CKD consider:

– monthly clinical review
– clinical assessment
  – blood pressure
  – weight
  – oedema
  – urine dipstick
– laboratory assessment
  – biochemical profile including urea, creatinine and electrolytes
  – eGFR
  – fasting glucose
  – fasting lipids
  – full blood count
  – iron stores
  – calcium and phosphate
  – parathyroid hormone (quarterly)
REFERRAL TO A NEPHROLOGIST

The CARI guidelines recommend that patients should be referred to a nephrologist at least 12 months prior to the anticipated commencement of dialysis and/or kidney transplantation (i.e. referral when eGFR < 30 mL/min/1.73 m²)\(^6\).

Appropriate referral is associated with:

– reduced rates of progression to end stage kidney disease
– decreased need for and duration of hospitalisation
– increased likelihood of permanent dialysis access created prior to dialysis onset
– reduced initial costs of care following the commencement of dialysis
– increased likelihood of kidney transplantation
– decreased patient morbidity and mortality

Clinical tip

When referring to a nephrologist ensure patient has had a recent kidney ultrasound, current blood chemistry, and quantification of proteinuria.

PRE-EMPTIVE TRANSPLANTATION

Pre-emptive transplantation means receiving a kidney transplant from a live donor prior to initiation of dialysis. The option of pre-emptive transplantation (transplantation prior to the initiation of dialysis) is associated with:\(^{24,25}\):

– reduced rates of death
– longer duration of functioning of the transplanted kidney
– psychosocial benefits
– economic benefits

A pre-emptive transplant can only be performed when the individual’s kidney function has deteriorated to a level that justifies the risks and complications of transplantation (eGFR usually 8–15 mL/min/1.73m²), but before they are so unwell that dialysis is essential.
CKD Management: Stage 5 (eGFR <15mL/min/1.73m²)

GOALS OF STAGE 5 CKD MANAGEMENT
- referral to a nephrologist for physical and psychosocial preparation for renal replacement therapy (dialysis, pre-emptive transplantation, transplantation) or conservative management
- reduction in cardiovascular and kidney risk
- early detection and management of complications
- avoidance of renally-excreted and nephrotoxic medications
- adjustment of medication doses to levels appropriate for kidney function

MONITORING IN STAGE 5 CKD
When monitoring patients with Stage 5 CKD consider:
- monthly clinical review (shared with renal unit)
- clinical assessment
  - blood pressure
  - weight
  - oedema
  - urine dipstick
- laboratory assessment
  - biochemical profile including urea, creatinine and electrolytes
  - eGFR
  - fasting glucose
  - fasting lipids
  - full blood count
  - iron stores
  - calcium and phosphate
  - parathyroid hormone (quarterly)
**Clinical tip**

While the renal unit undertakes most of the CKD management during this stage, it is still important that the patient maintains contact with their regular general practitioner to ensure coordination of whole-patient care, routine screening and health promotion, and psychosocial support.

**ADVANCED CARE DIRECTIVES**

During this stage it may be necessary to consider end of life decisions including advanced care directives to outline wishes for future health and personal care, including conservative treatment (no dialysis or transplantation), and palliative care arrangements.

The process varies between states, and more information is available from the Australian General Practice Network (www.agpn.com.au).
### Treatment targets for people with CKD\textsuperscript{17}

**Golden Rules**
- Blood pressure targets in CKD are $< 130/80$ mmHg or $< 125/75$ if proteinuria $> 1$ g/24hrs
- Urine protein:creatinine ratio of 100 mg/mmol $\approx$ daily protein excretion of 1g/24hrs
- Achieving adequate BP targets will often require the use of more than one agent
- As eGFR declines more drugs will typically be required to achieve target blood pressure

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Target</th>
<th>Treatment &amp; effects on systolic BP</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Lifestyle Factors</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smoking</td>
<td>Cease smoking</td>
<td>Lifestyle modification – refer to SNAP guide\textsuperscript{26}</td>
</tr>
<tr>
<td>Weight</td>
<td>BMI $\leq 25$ kg/m$^2$</td>
<td>Lifestyle modification – refer to SNAP guide</td>
</tr>
<tr>
<td></td>
<td>WC males $\leq 94$ cm$^2$ ($\leq 90$ cm in Asian populations)\textsuperscript{28}</td>
<td>SBP reduction $= 5–20$ mmHg/10 kg loss</td>
</tr>
<tr>
<td></td>
<td>WC females $\leq 80$ cm$^2$</td>
<td>SBP reduction $= 5–20$ mmHg/10 kg loss</td>
</tr>
<tr>
<td>Physical activity</td>
<td>$&gt; 30$ mins physical activity/day</td>
<td>Lifestyle modification – refer to SNAP guide</td>
</tr>
<tr>
<td></td>
<td>SBP reduction $= 4–9$ mmHg</td>
<td></td>
</tr>
<tr>
<td>Nutrition</td>
<td>Dietary salt intake 40–100 mmol/day\textsuperscript{29}</td>
<td>Lifestyle modification – refer to SNAP guide</td>
</tr>
<tr>
<td></td>
<td>SBP reduction $= 2–8$ mmHg</td>
<td></td>
</tr>
<tr>
<td>Alcohol</td>
<td>Moderate alcohol consumption only (1–2 standard drinks/day)</td>
<td>Lifestyle modification – refer to SNAP guide</td>
</tr>
<tr>
<td></td>
<td>SBP reduction $= 2–4$ mmHg</td>
<td></td>
</tr>
<tr>
<td><strong>Clinical Factors</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blood pressure</td>
<td>$&lt; 130/80$ mmHg</td>
<td>Lifestyle modification</td>
</tr>
<tr>
<td></td>
<td>$&lt; 125/75$ mmHg if proteinuria $&gt; 1$ g/24hrs</td>
<td>ACE inhibitor or ARB first-line</td>
</tr>
<tr>
<td>Proteinuria</td>
<td>$&gt; 50%$ reduction of baseline value</td>
<td>ACE inhibitor or ARB first-line</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>Total $&lt; 4.0$ mmol/L</td>
<td>Dietary advice</td>
</tr>
<tr>
<td></td>
<td>LDL $&lt; 2.5$ mmol/L</td>
<td>Statins</td>
</tr>
<tr>
<td>Blood glucose (for people with diabetes)</td>
<td>Pre-prandial BSL 4.4–6.7 mmol/L</td>
<td>Lifestyle modification</td>
</tr>
<tr>
<td></td>
<td>HbA1c $&lt; 7.0%$</td>
<td>Oral hypoglycaemics</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Insulin</td>
</tr>
</tbody>
</table>

The NHMRC also recommends immunisation against influenza and invasive pneumococcal disease for people with diabetes and/or end stage kidney disease.
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.

### Hypertension

**Target:**
- $< 130/80$ mmHg
- $< 125/75$ mmHg if proteinuria $> 1g/24$ hrs

**Urine protein:**
- Creatinine ratio of 100 mg/mmol
- Daily protein excretion of 1g/24 hrs

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 \(^{17,30,31}\).

**Management**

- **Lifestyle:** weight management, physical activity, limit alcohol intake to no more than two standard drinks per day (men), one standard drink per day (women), cease smoking, low salt diet
- **Multiple medications** (often three or more drugs) will be needed to control hypertension adequately in most patients with CKD \(^8\)
- People with diabetes or proteinuria should be treated with an ACE inhibitor or ARB as first line therapy \(^32\)
- When treatment with an ACE inhibitor or ARB is initiated, the creatinine and potassium levels can rise
  - If the acute rise in creatinine is less than 30% above the baseline level and stabilises within two months, the medication should be continued. People whose creatinine rises are most likely to achieve the greatest benefit in terms of kidney protection
  - If the rise in creatinine is greater than 30% above baseline value, the medication should be stopped and the person investigated for bilateral renal artery stenosis
  - If the serum potassium concentration is greater than 6 mmol/L despite dose reduction, diuretic therapy and dietary potassium restriction, then the medication (including spironolactone) should also be stopped \(^17\)
- **Diuretics** should be used in most patients \(^31\). Both nonloop diuretics (e.g. thiazides) and loop diuretics (e.g. frusemide) are effective as adjunct antihypertensive therapy. Additional agents can be chosen based on cardiovascular indications \(^31\)
- **Beta-blockers** may be useful in people with coronary heart disease, tachyarrhythmias and heart failure, but are contraindicated in asthma, chronic obstructive pulmonary disease and heart block
- Calcium channel blockers may be used for people with angina, the elderly and those with systolic hypertension
Principles of management of hypertension in people with CKD

Does the person have diabetes or proteinuria?

YES

Start ACEI or ARB

Monitor
  - eGFR
  - Potassium
  - Creatinine

NO

Stop ACEI/ARB

Continue to monitor BP and manage lifestyle risk factors

Does the person have diabetes or proteinuria?

YES

Decrease dose
  - Diuretic
  - Restrict dietary potassium

NO

Is BP at target < 130/80 (or < 125/75 if proteinuria > 1g)

YES

NO

Is potassium < 6mmol/L

YES

NO

Review antihypertensive medication and dose

Consider adding
  - Calcium channel blocker, or
  - Diuretic, or
  - Beta blocker

Increase ACEI or ARB

Person has CKD

Manage lifestyle risk factors continuously

Figure reproduced with permission from Med-E-Serv Pty Ltd, Chronic Kidney Disease (CKD) Update (http://www.kidney.primed.com.au/)
<table>
<thead>
<tr>
<th><strong>Lipids</strong></th>
<th>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 patients with proteinuria, particularly those with nephrotic syndrome. Dyslipidaemia should be treated as per cardiovascular disease recommendations and targets.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Target:</strong></td>
<td>- Dietary advice</td>
</tr>
<tr>
<td></td>
<td>- Statins (dose reduction not necessary)</td>
</tr>
<tr>
<td>Total &lt; 4.0 mmol/L</td>
<td></td>
</tr>
<tr>
<td>LDL &lt; 2.5 mmol/L</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Glycaemic Control</strong></th>
<th>Intensive blood glucose control significantly reduces the risk of developing microalbuminuria, macroalbuminuria and/or overt nephropathy in people with Type 1 and Type 2 diabetes.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Target:</strong></td>
<td>- Lifestyle modification</td>
</tr>
<tr>
<td></td>
<td>- Oral hypoglycaemics</td>
</tr>
<tr>
<td></td>
<td>- Insulin</td>
</tr>
<tr>
<td>Pre-prandial BSL 4.4-6.7 mmol/L</td>
<td></td>
</tr>
<tr>
<td>HbA1c &lt; 7.0%</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Proteinuria</strong></th>
<th>Proteinuria is an important prognostic feature of CKD. The degree of proteinuria relates to the severity of the kidney disease and with a greater likelihood of progression to end-stages of CKD. The amount of proteinuria can be reduced significantly by the use of an ACE inhibitor or ARB agent. Reduction in the amount of proteinuria is associated with improved outcomes.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Target</strong></td>
<td>- ACE inhibitor or ARB as first-line therapy</td>
</tr>
<tr>
<td></td>
<td>- Reduction in salt output through reducing oral salt intake</td>
</tr>
<tr>
<td></td>
<td>- Spironolactone (use with caution)</td>
</tr>
<tr>
<td>Proteinuria &gt; 50% reduction of baseline value after two to three months</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Sleep Apnoea</strong></th>
<th>Sleep apnoea can affect up to 50% of people with Stage 5 CKD.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Management</strong></td>
<td>- weight reduction</td>
</tr>
<tr>
<td></td>
<td>- avoid central nervous system depressants</td>
</tr>
<tr>
<td></td>
<td>- CPAP therapy (if obstructive pattern)</td>
</tr>
</tbody>
</table>
**Mineral and Bone Disorder**

**Target:**

PO$_4$ < 1.8 mmol/L  
PTH 2–4 x upper limit of normal

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. Calcium resorption by the kidney also decreases, leading to lower serum calcium levels. These two changes in turn stimulate parathyroid hormone excretion, which increases bone resorption.

In addition, as kidney function decreases the kidney produces less activated Vitamin D leading to increased prevalence of Vitamin D deficiency with declining renal function. These changes are associated with abnormal bone metabolism and increased risk of fracture and also increased cardiovascular mortality, perhaps mediated by accelerated vascular calcification.

**Management**

- **Phosphate**
  - Dietary restriction of phosphate. This is best managed with advice from a renal dietician
  - Use of phosphate binders. These are typically either calcium or non-calcium tablets taken with meals. These bind dietary phosphate to prevent absorption. The most common binder is Caltrate, available on PBS Authority for the indication of kidney disease and hyperphosphataemia

- **Calcium**
  - If phosphate is controlled, calcium will typically remain in normal range. If the level is low with normal phosphate level consider Vitamin D supplementation

- **Vitamin D**
  - Vitamin D may be used in CKD for two indications – Vitamin D deficiency and/or suppression of secondary hyperparathyroidism
  - Two agents are available for Vitamin D deficiency, either ergocalciferol or calcitriol. Ergocalciferol (Vitamin D2) is not the activated (Vitamin D3) form of the hormone, but can be used in early stage of CKD with Vitamin D deficiency
  - In later stages of CKD and/or for suppression of hyperparathyroidism, calcitriol is preferred. Calcitriol is available on PBS Authority for ‘the indication of hypocalcaemia due to renal disease’
  - Treatment to maintain PTH in desired range is via intervention above
  - The major side effect of therapy is hypercalcaemia
<table>
<thead>
<tr>
<th><strong>Anaemia</strong></th>
<th>Anaemia in CKD is related to both a reduction in erythropoietin production by the kidney and resistance to the action of erythropoietin. Anaemia related to CKD may occur at GFR of $\leq 60 \text{ mL/min/1.73m}^2$. The prevalence of anaemia increases with decreasing GFR.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Target:</strong></td>
<td>Hb $110 - 120 \text{ g/L}$</td>
</tr>
</tbody>
</table>

**Management**

- Other forms of anaemia should be excluded
- B12, folate and iron levels should be checked and corrected if deficient
- Thyroid stimulating hormone should be assessed and hypothyroidism treated if present
- Significant hyperparathyroidism or systemic inflammation may contribute to anaemia and also may cause refractoriness to erythropoietin therapy
- Treatment with erythropoietin is available for patients with anaemia related to CKD with GFR $< 60 \text{ mL/min/1.73m}^2$
- Treatment must be commenced by or in consultation with a nephrologist. There are three drugs currently available for this indication in Australia. All three are pre-filled syringes and are usually administered subcutaneously
- These drugs 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’\(^\text{38}\). A Private Hospital provider number is required to access the drug on Authority prescription
- Treatment can be divided into two phases:
  - Correction – treatment commenced with the aim of achieving target Hb. Monitor Hb two to four weekly and iron stores monthly. The aim is a rise of Hb at a rate of approximately 1 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 $110 - 120 \text{ g/L}$. There is evidence of harm when Hb exceeds $130 \text{ g/L}\(^\text{39}\). Monitoring of Hb and iron studies is generally at three monthly intervals
- Iron supplementation is typically required. This can be given either as oral iron or not uncommonly as intravenous supplementation
<table>
<thead>
<tr>
<th><strong>Dietary protein</strong></th>
<th>Dietary protein restriction has been shown to result in modest slowing of CKD progression(^4). However, the beneficial effect of protein restriction is typically outweighed by the deleterious effects of nutritional restriction.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Target:</strong></td>
<td>40 g/day</td>
</tr>
<tr>
<td>No lower than</td>
<td>0.75 g/kg body weight/day</td>
</tr>
<tr>
<td><strong>Management</strong></td>
<td></td>
</tr>
<tr>
<td>– dietary advice</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Malnutrition</strong></th>
<th>Poor food intake due to the symptoms of CKD can lead to malnutrition and low serum albumin.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Target:</strong></td>
<td>Serum albumin ≥ 35 g/L</td>
</tr>
<tr>
<td><strong>Management</strong></td>
<td></td>
</tr>
<tr>
<td>– dietary advice</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Uraemia</strong></th>
<th>Uraemia is a syndrome caused by the accumulation of the breakdown products of protein metabolism. The symptoms include anorexia, nausea, vomiting, lethargy, confusion, muscle twitching, convulsions and coma. Although urea and creatinine levels are high, the symptoms are most likely due to the accumulation of other toxic end products. These symptoms can lead to poor food intake and malnutrition. By the time uraemia becomes symptomatic dialysis is typically indicated.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Management</strong></td>
<td></td>
</tr>
<tr>
<td>– Dialysis should be commenced as soon as uraemic symptoms develop</td>
<td></td>
</tr>
<tr>
<td>– If dialysis is not planned:</td>
<td></td>
</tr>
<tr>
<td>– A low protein diet will help control gastrointestinal symptoms</td>
<td></td>
</tr>
<tr>
<td>– Fluid control should be strict to avoid pulmonary oedema</td>
<td></td>
</tr>
<tr>
<td>– Avoid unnecessary medications</td>
<td></td>
</tr>
<tr>
<td>– Anti-emetics are of limited value</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Acidosis</strong></th>
<th>People with stage 4–5 CKD 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.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Target:</strong></td>
<td>HCO(_3) &gt; 20 mmol/L</td>
</tr>
<tr>
<td><strong>Management</strong></td>
<td></td>
</tr>
<tr>
<td>– Supplementation with sodium bicarbonate (SodiBic) tablets to meet target</td>
<td></td>
</tr>
<tr>
<td>– Increased sodium load may worsen blood pressure control</td>
<td></td>
</tr>
</tbody>
</table>
| **Hyperkalaemia** | In CKD, excretion of potassium (K+) in the urine is impaired. Levels may also rise with ACE inhibitors and ARBs used to treat hypertension or with use of spironolactone. Levels consistently above 6.0 mmol/L are of concern and should be managed. Hyperkalaemia, especially levels > 6.5 mmol/L, predisposes to cardiac arrhythmias. **Management**  
- Low K+ diet  
- diuretics  
- resonium  
- cease ACE inhibitor/ARB if K+ persistently > 6.0 mmol/L, and not responsive to above therapies |
| **Target:**  
K+ ≤ 6.0 mmol/L | |
| **Restless Legs** | Restless Legs Syndrome (RLS) is common in CKD. As many as eight in ten people with Stage 5 CKD have RLS or a related movement disorder called periodic limb movements in sleep (PLMS).**Management**  
- iron replacement therapy  
- Dopaminergic agents or dopamine agonists (e.g. Levodopa, Ropinirole)  
- Benzodiazepines |
| **Sleep Apnoea** | Sleep apnoea can affect up to 50% of people with Stage 5 CKD.**Management**  
- weight reduction  
- avoid central nervous system depressants  
- CPAP therapy (if obstructive pattern) |
| **Depression** | Mental health problems, particularly depression, often occur with CKD. Depression is the most common mental health problem in people undergoing dialysis, affecting at least one-quarter to one-third of people 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.**Management**  
- Treatment of depressive symptoms in patients with chronic kidney disease has the potential to improve health outcomes.  
- Psychosocial interventions such as cognitive behavioural therapy, structured problem solving, interpersonal therapy and social work support can play a valuable role  
- There is good evidence for the use of antidepressants in treating depression in the context of chronic medical illness.  
- The medication should generally be introduced at a low dose which is then slowly increased |
The management of CKD is always a collaborative effort, involving at least the patient and their general practitioner. As kidney function declines, and as complications and co-morbidities increase, it becomes increasingly likely that the contribution of others will be needed for optimal care.

These may include:

- Family members or other lay carers
- Practice nurse
- Nephrologist
- Renal nurse/nurse practitioner
- Pharmacist
- Endocrinologist and other professionals specialising in diabetes
- Cardiologist
- Dietician
- Vascular and transplant surgeons
- Mental health professionals
- Community health professionals
- Social worker
- Aboriginal health worker

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

At times the general practitioner may be required to advocate for the patient with other professionals. In addition, he or she has continuing responsibility for primary care of the patient, including:

- Supporting and assisting the patient in the management of their kidney disease and other chronic health problems
- Responding appropriately to new symptoms
- Screening for developing problems and co-morbidities
- Provision of health promotion and disease prevention advice and interventions
- Assistance with addressing psychosocial issues
Even if the patient progresses to end stage kidney disease and has regular contact with the dialysis or transplant team, the general practitioner, practice staff and other health professionals remain vital to optimal care.

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

**USEFUL MBS ITEMS FOR CKD AND ITS COMPLICATIONS**

<table>
<thead>
<tr>
<th>For general practitioners</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>721</td>
<td>Preparation of a general practitioner management plan</td>
</tr>
<tr>
<td>723</td>
<td>Coordination of team care arrangements</td>
</tr>
<tr>
<td>725, 727, 729, 731</td>
<td>Contribution to or review of multidisciplinary care plans</td>
</tr>
<tr>
<td>734, 736, 738, 740, 742, 744, 746, 749, 757</td>
<td>Organisation and coordination of case conferences</td>
</tr>
<tr>
<td>759, 762, 765, 768, 771, 773, 775, 778, 779</td>
<td>Participation in case conferences</td>
</tr>
<tr>
<td>900, 903</td>
<td>Medication management review</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>For nephrologists</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>820, 822, 823, 830, 832, 834</td>
<td>Organisation and coordination of case conferences</td>
</tr>
<tr>
<td>825, 826, 828, 835, 837, 838</td>
<td>Participation in case conferences</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>For other health professionals</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>10950, 10951, 10954, 10956, 10958, 10968</td>
<td>Allied health services</td>
</tr>
</tbody>
</table>

Further information about these items is available at: www.health.gov.au/epc or contact your local Division of General Practice found at www.agpn.com.au
## Resources

For a full list of references contained in this document, please go to [www.kidney.org.au](http://www.kidney.org.au)

### Kidney Specific Resources

<table>
<thead>
<tr>
<th><strong>Kidney Health Australia</strong></th>
<th>Kidney Health Australia has a range of programs and services to assist general practitioners in their efforts to prevent, detect, and appropriately manage CKD:</th>
</tr>
</thead>
</table>
| [www.kidney.org.au](http://www.kidney.org.au) | - Health professional education programs  
- eGFR resources  
- Access to reports and publications  
- Patient fact sheets |
| **Kidney Check Australia Taskforce (KCAT)** | KCAT is Kidney Health Australia’s early detection and intervention program for health professionals. Initiatives include: |
| [www.kidney.org.au](http://www.kidney.org.au) | - Interactive workshops coordinated by Divisions of General Practice  
- Online learning programs  
- eGFR resources |
| **On-line learning programs** | KCAT have worked with PriMeD and Genesis Ed to develop a series of on-line CKD learning activities. |
- The Genesis Ed ‘The assessment and management of CKD’ program consists of a series of three one-hour e-chats which are now available to access online at [www.omnus.com.au](http://www.omnus.com.au)  
30 Category One, RACGP QA&CPD points can be earned by completing six hours of KCAT on-line education. |
<p>| <strong>Caring for Australasians with Renal Impairment (CARI)</strong> | Online evidence-based clinical practice guidelines for the management of adult and paediatric patients with chronic kidney disease. |</p>
<table>
<thead>
<tr>
<th><a href="http://www.cari.org.au">www.cari.org.au</a></th>
<th></th>
</tr>
</thead>
</table>

**General Practice Resources**

| **Royal Australian College of General Practitioners**<br>www.racgp.org.au | Professional guides available to download online including:  
– Smoking, Nutrition, Alcohol and Physical Activity (SNAP) Guide  
– Red Book: Guidelines for preventive activities in general practice  
– Green Book: Putting prevention into practice  
– National guide to a preventive assessment in Aboriginal and Torres Strait Islander peoples |
|-----------------------------|--------------------------------------------------------------------------------------------------|

| **Australian General Practice Network**<br>www.agpn.com.au | The peak national body representing divisions of general practice and their state-based organisations across Australia. Online resources include:  
– Chronic Disease Management  
– Lifescrpts  
– Enhanced Divisional Quality Use of Medicines |
|-----------------------------|--------------------------------------------------------------------------------------------------|

<table>
<thead>
<tr>
<th><strong>Type 2 Diabetes from the GP’s perspective</strong>&lt;br&gt;www.servier.com.au</th>
<th>NEFRON study data coupled with pragmatic advice for general practitioners</th>
</tr>
</thead>
</table>
## Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACE inhibitor</td>
<td>Angiotensin-Converting Enzyme inhibitor</td>
</tr>
<tr>
<td>ARB</td>
<td>Angiotensin II Receptor Blocker</td>
</tr>
<tr>
<td>CARI</td>
<td>Caring for Australasians with Renal Impairment</td>
</tr>
<tr>
<td>BMI</td>
<td>body mass index</td>
</tr>
<tr>
<td>CKD</td>
<td>chronic kidney disease</td>
</tr>
<tr>
<td>CVD</td>
<td>cardiovascular disease</td>
</tr>
<tr>
<td>eGFR</td>
<td>estimated glomerular filtration rate</td>
</tr>
<tr>
<td>FBC</td>
<td>full blood count</td>
</tr>
<tr>
<td>GFR</td>
<td>glomerular filtration rate</td>
</tr>
<tr>
<td>MDRD</td>
<td>Modification of Diet in Renal Disease</td>
</tr>
<tr>
<td>NSAIDS</td>
<td>non-steroidal anti-inflammatory drugs</td>
</tr>
<tr>
<td>PBS</td>
<td>Pharmaceutical Benefits Scheme</td>
</tr>
<tr>
<td>PTH</td>
<td>parathyroid hormone</td>
</tr>
<tr>
<td>WC</td>
<td>waist circumference</td>
</tr>
</tbody>
</table>
Acknowledgements
Kidney Health Australia and the Kidney Check Australia Taskforce acknowledges with thanks those who contributed to the development of this document. In particular, thanks are due to the Editorial Advisory Group members as follows:

Prof Steve Chadban
Ms Barb Harvie
Dr Beres Joyner
Dr Paul Snelling
Prof Tim Usherwood
Dr Andrew Weekes
Dr Sheena Wilmot
A/Prof Tim Mathew
(Kidney Health Australia)
Dr Marie Ludlow
(Kidney Health Australia)

Citation: Chronic Kidney Disease (CKD) Management in General Practice. Kidney Health Australia, Melbourne, 2007.

The content of this booklet has been endorsed by the Royal Australian College of General Practitioners and the Australian and New Zealand Society of Nephrology.

An electronic version of this booklet is available at www.kidney.org.au or www.racgp.org.au

© Kidney Health Australia
This report is copyright. Apart from any use as permitted under the Copyright Act 1968, no part may be reproduced without written permission from Kidney Health Australia. Requests and enquiries concerning reproduction and rights should be directed to Kidney Health Australia, PO Box 9993, Melbourne VIC 3001.

ISBN: 978-0-9803160-6-3

Published by
Kidney Health Australia, 2007.
Reprinted 2010.

A Summary Guide of this booklet "Chronic Kidney Disease (CKD) Management in General Practice" may be downloaded free of charge from the Health Professionals section of the Kidney Health Australia website at www.kidney.org.au.

Disclaimer
This guide 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 healthcare professional making use of this guide is responsible for evaluating 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.

Kidney Health Australia and the Kidney Check Australia Taskforce would like to acknowledge our sponsor Servier Laboratories who provided an unrestricted educational grant for this initiative.
# CKD management according to stage

<table>
<thead>
<tr>
<th>CKD Stage</th>
<th>Description</th>
<th>eGFR (mL/min/1.73m²)</th>
<th>Common Signs and Symptoms</th>
<th>Common Complications</th>
<th>Clinic Assessment</th>
<th>Lab Assessment</th>
<th>Management</th>
<th>Frequency of clinical review</th>
<th>Nephrologist Referral</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Kidney damage + normal or ↑ eGFR</td>
<td>≥ 90</td>
<td>Nil</td>
<td>Hypertension</td>
<td>BP, Weight, Urine dipstick</td>
<td>General chemistry, eGFR, Glucose, Lipids</td>
<td>Diagnosis Cardiac and kidney risk factor modification Treat BP to target &lt; 130/80 mmHg or &lt; 125/75 mmHg if proteinuria &gt; 1g/24hrs (urine protein: creatinine ratio of 100 mg/mmol = daily protein excretion of 1g/24hrs)</td>
<td>4–6 monthly</td>
<td>Consider referral if indication is present</td>
</tr>
<tr>
<td>2</td>
<td>Kidney damage + mild ↓ eGFR</td>
<td>60 – 89</td>
<td>Nil or nocturia, mild malaise, anorexia</td>
<td>As for stage 1–2 + Mineral and Bone Disorder Anaemia Sleep Apnoea Restless legs CVD Malnutrition Depression</td>
<td>As for stage 1-2</td>
<td>As for stage 1-2 + FBC Iron stores Ca/PO₄ PTH (quarterly)</td>
<td>As per stage 1–2 + treat complications Medication review</td>
<td>1–3 monthly</td>
<td>Consider referral if indication is present</td>
</tr>
<tr>
<td>3</td>
<td>Moderate ↓ eGFR</td>
<td>30–59</td>
<td>As for stage 3 + nausea, pruritis, restless legs, dyspnoea</td>
<td>As for stage 3 + Hyperphosphataemia Acidosis Hyperkalaemia</td>
<td>As for stage 1–2 + Oedema</td>
<td>As for stage 3</td>
<td>As for stage 3 + Dialysis education Dialysis access surgery</td>
<td>Monthly</td>
<td>All patients should be referred to a nephrologist</td>
</tr>
<tr>
<td>4</td>
<td>Severe ↓ eGFR</td>
<td>15–29</td>
<td>As for stage 4</td>
<td>As for stage 3 + Pericarditis GIT bleeding Encephalopathy Neuropathy</td>
<td>As for stage 4</td>
<td>As per monthly blood schedule specified by Renal Unit</td>
<td>As for stage 4 + Dialysis or transplantation (or conservative medical management)</td>
<td>Monthly (shared with renal unit)</td>
<td>All patients should be referred to a nephrologist</td>
</tr>
<tr>
<td>5</td>
<td>End stage kidney disease</td>
<td>&lt;15 or on dialysis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>