Management of Stage 3 Chronic Kidney Disease in General Practice

KCAT Primary Care Workshop

This workshop was conceived and developed by the Kidney Check Australia Taskforce with particular thanks to Dr Paul Snelling

V0914
KCAT Supporters

The KCAT program is proudly supported by unrestricted educational grants from:

KCAT Program Partners

KCAT Major Sponsor
Learning outcomes
At the end of this presentation participants will be able to:

- Demonstrate the ability to stage chronic kidney disease (CKD) through accurate interpretation of kidney function
- Define the goals for best practice management of CKD, particularly Stage 3
- Determine when to refer patients with CKD to a Nephrologist according to the recommended clinical indicators
- Implement a practice-based system, for patient safety, to identify patients at higher risk of CKD for a kidney health check
What is CKD?

Chronic kidney disease is defined as:

Glomerular Filtration Rate (GFR) < 60 mL/min/1.73m² for ≥3 months with or without evidence of kidney damage.

OR

Evidence of kidney damage (with or without decreased GFR) for ≥3 months:

- albuminuria
- haematuria after exclusion of urological causes
- pathological abnormalities
- anatomical abnormalities.

Kidney disease in Australia

Australians aged ≥ 18 years

5+ MILLION AT RISK

591,000

Stage 3 CKD

Stage 1 - 2 CKD

Stage 4 - 5 CKD

Dialysis or transplant

Hypertension / Diabetes

Less than 10% of these people are aware they have CKD

Australian Health Survey 2013; ABS population estimates June 2013; ANZDATA 2012 Report

CKD staging is according to the CKD-EPI equation.
Staging CKD

Combine eGFR stage, albuminuria stage and underlying diagnosis to specify CKD stage
(e.g. stage 3b CKD with microalbuminuria secondary to diabetic kidney disease)

<table>
<thead>
<tr>
<th>GFR Stage</th>
<th>GFR (mL/min/1.73m²)</th>
<th>Albuminuria Stage</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Normal (urine ACR mg/mmol)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Male: &lt; 2.5 Female: &lt; 3.5</td>
</tr>
<tr>
<td>1</td>
<td>≥90</td>
<td>Not CKD unless haematuria, structural or pathological abnormalities present</td>
</tr>
<tr>
<td>2</td>
<td>60-89</td>
<td></td>
</tr>
<tr>
<td>3a</td>
<td>45-59</td>
<td></td>
</tr>
<tr>
<td>3b</td>
<td>30-44</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>15-29</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>&lt;15 or on dialysis</td>
<td></td>
</tr>
</tbody>
</table>

Colour-coded Clinical Action Plans

Staging CKD

‘CKD Management in General Practice’ booklet uses colour-coded action plans indicating the overall risk of

– Progression of CKD
– Cardiovascular events
Why worry about CKD & ESKD?

Figure 3.1: Prevalence of registered and projected treated-ESKD, for all patients and by incident age, 2003–2020

Notes
1. The solid lines represent existing registry data while the dotted lines are for projected prevalent cases.
2. The percentage increases from 2011 to 2020 for each age group and for total cases are listed in the legends.

Source: AIHW analysis of ANZDATA Registry data.
CKD survival

5 year survival of patients aged 60 years with common cancers compared with CKD

% of 5 Year Survival

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Testicular</th>
<th>Breast</th>
<th>Bladder</th>
<th>Kidney Transplant</th>
<th>Rectal</th>
<th>Cervical</th>
<th>Colon</th>
<th>Stage 5 CKD on dialysis</th>
<th>Ovarian</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>95</td>
<td>85</td>
<td>75</td>
<td>75</td>
<td>62</td>
<td>60</td>
<td>54</td>
<td>46</td>
<td>44</td>
</tr>
</tbody>
</table>
ESKD and cardiovascular mortality

Dialysis patients

General population

Annual Mortality (%)
Intervention and management

GFR (ml/min/1.73 m²)

Effective Interventions

Residual Renal Function

Late Referral

Timely Start?
CVD risk in CKD

Kidney & cardiovascular outcomes in patients with CKD

*Kaiser Permanente Longitudinal Study*

Patients with CKD are 20 times more likely to die from cardiovascular events than survive to reach dialysis

Keith et al Arch Int Med 164 (6) 659, 2004
Age and kidneys

Relationship between age and kidney function

Relationship of eGFR to age

Australasian Creatinine Consensus group. MJA 2007; 187(8): 459-463
Case Study - Bruce B.

Background
- 74 years old
- Retired small business owner
# Case study - Bruce

## Medical History

<table>
<thead>
<tr>
<th></th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Smoker</strong></td>
<td>20 cigarettes/day (35 pack-year history)</td>
</tr>
<tr>
<td><strong>Alcohol</strong></td>
<td>30g/day (3 standard drinks)</td>
</tr>
<tr>
<td><strong>Hypertension</strong></td>
<td>20 years</td>
</tr>
<tr>
<td><strong>Type 2 Diabetes</strong></td>
<td>5 years - <em>takes oral hypoglycaemics</em></td>
</tr>
<tr>
<td><strong>Infra-renal AAA 4cm</strong></td>
<td>Incidental finding on CT for abdominal pain</td>
</tr>
<tr>
<td><strong>Stress echo</strong></td>
<td>No inducible ischaemia</td>
</tr>
<tr>
<td><strong>Medications</strong></td>
<td>Amlodopine, Pravastatin, Gliclazide, Aspirin (low dose)</td>
</tr>
</tbody>
</table>
## Case study – today’s visit

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood Pressure</td>
<td>190 / 84 mmHg</td>
</tr>
<tr>
<td></td>
<td>Peripheral pulses present</td>
</tr>
<tr>
<td>Creatinine</td>
<td>160 µmol/L</td>
</tr>
<tr>
<td>eGFR</td>
<td>36 mL/min/1.73m² (has been consistently</td>
</tr>
<tr>
<td></td>
<td>below 40 mL/min/1.73m² for 6 months)</td>
</tr>
<tr>
<td>Total cholesterol</td>
<td>6.7 mmol/L</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>4.05 mmol/L</td>
</tr>
<tr>
<td>Full Blood Count</td>
<td>Normal</td>
</tr>
<tr>
<td>Urine ACR</td>
<td>2.4 mg/mmol</td>
</tr>
<tr>
<td>Fundi</td>
<td>Normal</td>
</tr>
</tbody>
</table>
## Case study - Bruce

<table>
<thead>
<tr>
<th>GFR 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></td>
<td></td>
<td>Male: &lt; 2.5</td>
<td>Female: &lt; 3.5</td>
<td>Male: 2.5-25</td>
</tr>
<tr>
<td>1</td>
<td>≥90</td>
<td>Not CKD unless haematuria, structural or pathological abnormalities present</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>60-89</td>
<td></td>
<td></td>
<td></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>BRUCE’S RESULTS</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>PUT HIM HERE</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>
<td></td>
</tr>
</tbody>
</table>

Case study – True or False

Answer True or False to each of the statements below

a) The absence of albuminuria excludes diabetic kidney disease

b) Quantitation of albuminuria will give important prognostic information

c) He should not be started on an Angiotensin converting enzyme (ACE) inhibitors and angiotensin II receptor blockers (ARB) to slow progression of kidney disease as he has CKD Stage 3b

d) His smoking will worsen his kidney function

e) Lipid lowering therapy has been proven to slow progression of kidney disease

f) Cardiovascular disease risk – Should Bruce's CVD be determined using the absolute risk tool?
Case study - Answer

a) The absence of albuminuria excludes diabetic kidney disease

FALSE

– 20-30% of diabetic patients may have chronic kidney disease without evidence of albuminuria

⇒ **Mechanism not well understood**

– Likely to progress with time
Case study - Answer

b) Quantitation of albuminuria will give important prognostic information

TRUE

- Increasing degrees of albuminuria lead to increasing risk of ESKD
  ⇒  *Albuminuria is a stronger marker of risk of progression to ESKD than baseline eGFR*
  ⇒  *But eGFR strong predictor of morbidity and mortality*

- Reduction of albuminuria predicts reduced mortality and reduced progression to ESKD
CKD risk

eGFR <60mL/min and Urine ACR >1.1 mg/mmol are independent predictors of mortality in the general population

- Meta analysis
- 105 872 participants
- 730 577 person-years
- 14 studies

CKD prognosis consortium; Lancet 2010
Kidney function marker

Macroalbuminuria is a better marker than GFR in predicting loss of kidney function

N=8952 – F/U 4yrs
Albuminuria & ↓ GFR predict mortality & morbidity (RR)

<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>Macroalbuminuria</th>
<th>↓GFR</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mortality (RR)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CV</td>
<td>1</td>
<td>2.6</td>
<td>3.4</td>
</tr>
<tr>
<td>non CV</td>
<td>1</td>
<td>1.5</td>
<td>3.0</td>
</tr>
<tr>
<td><strong>Morbidity (RR)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CV</td>
<td>1</td>
<td>1.4</td>
<td>2.3</td>
</tr>
</tbody>
</table>

PREVEND Study; J Am Soc Nephrol 2006
Case study - Answer

c) He should not be started on an ACE inhibitor or ARB to slow progression of kidney disease as he has CKD Stage 3b

FALSE

– ACEi’s and ARB’s have been shown to reduce the risk of CV events and death in people with CKD
Treatment of BP in CKD

- ACE inhibitor (ACEi) or Angiotensin Receptor Blocker (ARB)
- CKD patients often need multiple medications to achieve BP control
- Goal is to maintain BP consistently below target

Bakris et al, AJKD 2000;36:646-661
Intervention and outcomes
ACE inhibitors in Type 2 Diabetes with hypertension
The BENEDICT Trial

Adjusted Health Risks (HR) for major cardiovascular events according to baseline albuminuria

Risk of CVD is significantly reduced

Ruggenenti et al; JASN 2012
## Target blood pressure in adults

### Blood Pressure goals

<table>
<thead>
<tr>
<th>Patient Group</th>
<th>Maintain BP consistently</th>
<th>Blood Pressure Goal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Albuminuria</td>
<td>&lt;130/80</td>
<td>(mmHg)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>&lt;130/80</td>
<td></td>
</tr>
<tr>
<td>Chronic Kidney Disease</td>
<td>&lt;140/90</td>
<td></td>
</tr>
</tbody>
</table>
Risk stratification – Blood pressure

- 10 mmHg ↑ Systolic Blood Pressure results in 10.9% increase in Relative Risk of ESKD (RENAAL Study*)

- Greatest reduction in mortality in those with Pulse Pressure > 90 mmHg in RENAAL*

RENAAL Study, Keane et al 2003; 63: 1499-1507
Q1d) His smoking will worsen his kidney function

TRUE

- Smoking is associated with kidney damage in the population AusDiab Study
- Smoking increases proteinuria and accelerates loss of GFR

* p<0.05 compared with ‘never smokers’

Adjusted for age, education, physical activity, diabetes, CVD, BP medication, systolic BP, lipids, WC, eGFR & ACR

Hallan & Orth; Kidney Int 2011
Case study - Answer

e) Lipid lowering therapy has been proven to slow progression of kidney function

FALSE

- No specific randomised trials
- Post hoc analysis of CKD trials show no consistent pattern of responses
- No trials in GFR <40 mL/min/1.73m²
Case study - Answer

f) Assessment for cardiovascular disease risk - Should Bruce's CVD be determined using the absolute risk tool?

FALSE

- People with moderate or severe CKD (defined as persistently having a urine ACR>25mg/mmol (males) or > 35mg/mmol (females) or eGFR <45ml/min/1.73m² are considered to be at the highest risk of a cardiovascular event and do not need to be assessed by the cardiovascular risk tool.
Case study - Answer

e) Lipid lowering therapy has been proven to slow progression of kidney function

FALSE

- No specific randomised trials
- Post hoc analysis of CKD trials show no consistent pattern of responses
- No trials in GFR <40 mL/min/1.73m²
Lipid lowering in CKD

Study of Heart And Renal Protection (SHARP)

There is strong evidence that lipid lowering in people with CKD will decrease the risk of atherosclerotic events

• Recruited 9438 patients with CKD, including 1/3 on dialysis
• No previous cardiovascular events
• Randomised to 20 mg simvastatin + 10 mg ezetimibe vs placebo
• Mean baseline LDL-C level of 2.8mmol/L
• Outcome was first major atherosclerotic event (non-fatal myocardial infarction or coronary death, non-haemorrhagic stroke, or any arterial revascularisation procedure)
• Followed for an average of 5 yrs

Baigent et al, Lancet 2011
CV events

SHARP results: 17% reduction in major atherosclerotic events*

Risk ratio 0.83 (0.74 – 0.94)
Log rank p=0.0022

17% reduction in risk

Placebo
Eze/simv

*Major atherosclerotic events (coronary death, MI, non-haemorrhagic stroke, or any revascularization)

*Average 0.85mmol/L decrease in LDL-C vs. placebo

Baigent et al, Lancet 2011
Case study - Bruce

- Commenced on perindopril / indapamide
- Usual practice is to increase by one antihypertensive at a time
- Seen 4 weeks later and reassessed

<table>
<thead>
<tr>
<th></th>
<th>4 Weeks ago</th>
<th>Today</th>
</tr>
</thead>
<tbody>
<tr>
<td>BP</td>
<td>190/84 mmHg</td>
<td>150/76 mmHg</td>
</tr>
<tr>
<td>Creatinine</td>
<td>160 μmol/L</td>
<td>189 μmol/L</td>
</tr>
<tr>
<td>eGFR</td>
<td>36 mL/min/1.73m²</td>
<td>29 mL/min/1.73m²</td>
</tr>
<tr>
<td>Potassium</td>
<td>-</td>
<td>5.8 mmol/L</td>
</tr>
<tr>
<td>Urine ACR</td>
<td>2.4 mg/mmol</td>
<td>2.6 mg/mmol</td>
</tr>
</tbody>
</table>
Case study - Question

Do You...

a) Cease the ACEi and commence another drug
b) Cease the ACEi and check for a renal artery stenosis
c) Continue the ACEi and check for a renal artery stenosis
d) Add another drug for better BP control
Case study – Answer

a) Add another drug for better blood pressure control

Rationale:
• A decrease in eGFR of <25% is not unexpected after BP lowering and is a result of decreased perfusion
• Target BP in CKD is <140/90 mmHg (130/80 if albuminuria present)
• ACEi may have particular benefit for kidney disease
• K+ needs watching but not an immediate concern at this level
• give advice regarding low K+ diet
### Case study - Bruce

**Seen 1 month later**

<table>
<thead>
<tr>
<th></th>
<th>1st Visit</th>
<th>4 wks later</th>
<th>Today (8 wks later)</th>
</tr>
</thead>
<tbody>
<tr>
<td>BP</td>
<td>190/84 mmHg</td>
<td>150/76 mmHg</td>
<td>134/68 mmHg</td>
</tr>
<tr>
<td>Creatinine</td>
<td>160 µmol/L</td>
<td>189 µmol/L</td>
<td>245 µmol/L</td>
</tr>
<tr>
<td>eGFR</td>
<td>36 mL/min/1.73m²</td>
<td>29 mL/min/1.73m²</td>
<td>22 mL/min/1.73m²</td>
</tr>
<tr>
<td>Potassium</td>
<td>-</td>
<td>5.8 mmol/L</td>
<td>5.4 mmol/L</td>
</tr>
<tr>
<td>Ca</td>
<td></td>
<td></td>
<td>2.05 mmol/L</td>
</tr>
<tr>
<td>PO₄</td>
<td></td>
<td></td>
<td>1.54 mmol/L</td>
</tr>
<tr>
<td>Hb</td>
<td></td>
<td></td>
<td>98g/L Normocytic / normochromic</td>
</tr>
<tr>
<td>Urine ACR</td>
<td>2.4 mg/mmol</td>
<td>2.6 mg/mmol</td>
<td>2.6 mg/mmol</td>
</tr>
</tbody>
</table>
Case study - Question

What would you do?

a) Check iron studies
b) Check Vit B12 and folate levels
c) Check Vitamin D and PTH
d) Continue ACE inhibitor
e) All of the above
### Results

<table>
<thead>
<tr>
<th>Test</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ferritin</td>
<td>350 mg/L Tsat 55%</td>
</tr>
<tr>
<td>B12 and Folate</td>
<td>Normal</td>
</tr>
<tr>
<td>TSH</td>
<td>Normal</td>
</tr>
<tr>
<td>PTH</td>
<td>18 pmol/L (N&lt;8 pmol/L)</td>
</tr>
<tr>
<td>Vitamin D</td>
<td>25 nmol/L - moderate deficiency</td>
</tr>
</tbody>
</table>

**Case study - Answer**

**e. All of the above**
Anaemia

• Anaemia of CKD is related to both:
  - reduced erythropoietin production by the kidney
  - resistance to the action of erythropoietin

• Anaemia due to CKD begins at GFR < 60 mL/min
  - Prevalence of anaemia increases markedly with decreasing GFR
  - Common when GFR < 30 mL/min (30-40%)

• **CKD anaemia is a diagnosis of exclusion**
  - Need to ensure not Fe deficient or B12/ folate deficient, or hypothyroid

Gouva et al; Kidney Int 2004
Anaemia is associated with mortality in dialysis patients

Adjusted Relative Risk of death due to any cardiac cause, according to Haematocrit

Randomised trials have not shown a benefit and have shown some harm. Individualise treatment per patient and refer to PBS criteria.

Li & Collins; Kidney International 2004
The target Hb for anaemia in CKD

Optimal Hb level not known

RCT – no benefit above 120 g/L

(?harm if Hb rises quickly)

Individualise treatment

Refer to PBS criteria
CKD & anaemia summary

- Common and important to correct
- PBS criteria - can’t start EPO till Hb <100g/L
- Need to have Nephrologist endorsement to start
- Ensure not iron deficient
- All respond – need to dose titrate
- Most self administer SC every 1, 2 or 4wks (depending on EPO formulation)
- All will need extra iron (oral or i.v.)
Case study - Bruce

Calcium and phosphate at today’s visit

<table>
<thead>
<tr>
<th></th>
<th>1st Visit</th>
<th>4 wks later</th>
<th>Today ( 8 wks later)</th>
</tr>
</thead>
<tbody>
<tr>
<td>BP</td>
<td>190/84 mmHg</td>
<td>150/76 mmHg</td>
<td>134/68 mmHg</td>
</tr>
<tr>
<td>Creatinine</td>
<td>160 µmol/L</td>
<td>189 µmol/L</td>
<td>245 µmol/L</td>
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<td>eGFR</td>
<td>37 mL/min/1.73m²</td>
<td>29 mL/min/1.73m²</td>
<td>22 mL/min/1.73m²</td>
</tr>
<tr>
<td>Urine ACR</td>
<td>2.4 mg/mmol</td>
<td>2.6 mg/mmol</td>
<td>2.6 mg/mmol</td>
</tr>
<tr>
<td>Ca</td>
<td></td>
<td></td>
<td><strong>2.05 mmol/L</strong></td>
</tr>
<tr>
<td>PO₄</td>
<td></td>
<td></td>
<td><strong>1.54 mmol/L</strong></td>
</tr>
<tr>
<td>Hb</td>
<td></td>
<td></td>
<td>98g/L Normocytic / normochromic</td>
</tr>
</tbody>
</table>
Complications – mineral and bone disorder

- Changes in metabolism of calcium, phosphate, parathyroid hormone and Vitamin D common when eGFR ≤60 mL/min/1.73m²

- Leads to:
  - Bone disease
  - Soft tissue calcification (coronaries & valves)
  - Pruritus
  - Proximal myopathy
  - Premature death
Phosphate

Increased $\text{PO}_4$ associated with increased mortality even in normal kidney function

Tonelli et al, Circulation 2006
Mechanisms of Ca/PO$_4$ disturbance

1. **Phosphate retention** with reduced GFR results in increased serum PO$_4$ and suppresses Vitamin D3 production

2. **Reduced Vitamin D3** leads to reduced Ca absorption and this plus high serum PO$_4$ leads to **low serum Calcium**

3. Ca x PO$_4$ increases favouring tissue deposition

4. PTH stimulated by low Ca, high PO$_4$ & low Vit D3

**Clinical effects:**
- Low serum Calcium
- High serum Phosphate
- High serum PTH
- Low Vitamin D3 [$1,25 \text{(OH)}_2\text{D}_3 = \text{calcitriol}$]
## Changes with reducing GFR

<table>
<thead>
<tr>
<th>GFR (mL/min/1.73m$^2$)</th>
<th>CKD Stage</th>
<th>Changes in serum levels</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>1,25D</td>
</tr>
<tr>
<td>60-90</td>
<td>2</td>
<td>↓</td>
</tr>
<tr>
<td>30-59</td>
<td>3</td>
<td>↓↓</td>
</tr>
<tr>
<td>15-30</td>
<td>4</td>
<td>↓↓</td>
</tr>
<tr>
<td>&lt;15</td>
<td>5</td>
<td>↓↓</td>
</tr>
</tbody>
</table>
### Assessment of Ca/PO$_4$ disturbance

*(CKD Mineral and Bone disorder)*

#### What to Measure & How Often

<table>
<thead>
<tr>
<th>Test</th>
<th>Progressive CKD stage 3</th>
<th>CKD stage 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calcium &amp; phosphate</td>
<td>6-12 months</td>
<td>3-6 months</td>
</tr>
<tr>
<td>PTH &amp; alkaline phosphatase</td>
<td>Baseline</td>
<td>6-12 months</td>
</tr>
<tr>
<td>25-hydroxyvitamin D</td>
<td>Baseline</td>
<td>Baseline</td>
</tr>
</tbody>
</table>
## Goals of therapy for mineral and bone disorder

<table>
<thead>
<tr>
<th>GOALS OF THERAPY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Keep PO₄ in normal range (0.8-1.5 mmol/L)</td>
</tr>
<tr>
<td>Keep Ca in normal range (2.2-2.6 mmol/L)</td>
</tr>
<tr>
<td>Keep PTH 2-9 x upper limit of normal and avoid trends towards the extremes of this range</td>
</tr>
<tr>
<td>25-hydroxyvitamin D optimal levels may be &gt; 75 nmol/L</td>
</tr>
</tbody>
</table>
## Therapy for Ca/PO$_4$ disturbance

<table>
<thead>
<tr>
<th>THERAPY FOR Ca/Po$_4$ DISTURBANCE</th>
</tr>
</thead>
<tbody>
<tr>
<td>✧ Control sPO$_4$</td>
</tr>
<tr>
<td>Dietary Restriction</td>
</tr>
<tr>
<td>Phosphate binders (prevent uptake)</td>
</tr>
</tbody>
</table>

|✧ Control sCa                     |
|Adequate calcium uptake           |
|Calcitrol (increases uptake)      |

|✧ Control sPTH                    |
|Calcitrol                         |
|Cinacalcet                        |
|Parathyroidectomy                 |
Referral is recommended if:

- eGFR <30mL/min/1.73m²
- Persistent significant albuminuria (urine ACR ≥ 30mg/mmol)
- Rapidly declining eGFR from a baseline of <60 mL/min/1.73m² (a decline of >5mL/min/1.73m² over a six-month period which is confirmed on at least three separate readings)
- CKD and hypertension that is hard to get to target despite at least three anti-hypertensive agents

Clinical tip
When referring to a Nephrologist ensure patient has had a recent urine ACR, current blood chemistry and haematology and a urinary tract ultrasound and urine MCS.

Anyone with an acute presentation and signs of acute nephritis (oliguria, haematuria, acute hypertension, and oedema) should be regarded as a medical emergency and referred without delay.
Referral is NOT usually necessary if:

- Stable eGFR ≥30 mL/min/1.73m²
- Urine ACR < 30mg/mmol (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.

Useful Tips

- Pay attention to CVD risk reduction
- Consider discussing management issues with a Nephrologist in cases where uncertainty regarding referral exists.
- Don’t refer to Nephrologist if targets of therapy are achieved
- Spiral CT angiogram for hypertension is not recommended without specialty advice
Managing CKD

Progressive CKD is often associated with:

- Depression
- Macular degeneration
- Impaired cognitive function
## Conclusion

- Early CKD is common and can be primarily managed in general practice
- Therapy overlaps significantly with best practice in CV risk reduction and diabetes care

## Key CKD management tasks

<table>
<thead>
<tr>
<th>Task</th>
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<tbody>
<tr>
<td>Lifestyle – Healthy diet &amp; exercise, no smoking, weight control</td>
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<tr>
<td>Reduce CV risk</td>
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<tr>
<td>BP at target with ACEi or ARB and other agents as required</td>
</tr>
<tr>
<td>Reduce albuminuria with ACEi or ARB</td>
</tr>
<tr>
<td>Optimise haemoglobin, Ca/P and glycaemia</td>
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</tbody>
</table>
Resources

CKD Management in General Practice
2012 Guidelines booklet

Available at www.kcat.org.au
Resources

CKD Patient fact sheets
Available along with more kidney health fact sheets at
www.kidney.org.au > For Patients > Health Fact Sheets
Resources

Kidney Health Information Service

Free call information service for people living with / affected by kidney disease
Join the Kidney Community

KIDNEY COMMUNITY members receive a monthly newsletter from KHA allowing you to access:

- Information and invitations to KHA's education and support activities
- Updates on medical research in kidney disease
- Updates on clinical trials and research opportunities
- Information on advocacy opportunities and government relations issues
- Information on community and corporate events held by Kidney Health Australia

To join the kidney community, email community@kidney.org.au
Questions?

Thankyou for participating in this workshop

*Please complete your evaluation form before leaving.*