

# CHRONIC KIDNEY DISEASE (CKD)

## MANAGEMENT IN GENERAL PRACTICE



GUIDANCE AND CLINICAL TIPS TO HELP IDENTIFY,  
MANAGE AND REFER **CKD** IN YOUR PRACTICE



2nd edition 2012  
[www.kidney.org.au](http://www.kidney.org.au)



## Key Clinical Tips

A measured or estimated GFR  $< 60$  mL/min/1.73 m<sup>2</sup> is associated with increased risks of adverse renal, cardiovascular and other clinical outcomes, irrespective of age.

For people with CKD, the combination of low GFR and albuminuria places them at greater risk of CKD and CVD progression at all ages than those with just one of low GFR or albuminuria.

The preferred method for assessment of albuminuria in both diabetes and non-diabetes is urinary ACR measurement in a first void spot specimen. Where a first void specimen is not possible or practical, a random spot urine specimen for urine ACR is acceptable.

People with CKD should be treated with blood-pressure lowering drugs to maintain a blood pressure that is consistently below 140/90 mmHg. If albuminuria is present (urine ACR  $>3.5$  mg/mmol in females and  $>2.5$  mg/mmol in males) a consistent blood pressure below 130/80 mmHg should be achieved.

If diabetes is present, the blood pressure should be consistently maintained below 130/80 mmHg.

Citation: Chronic Kidney Disease (CKD) Management in General Practice (2nd edition). Kidney Health Australia, Melbourne, 2012.

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

An electronic version of this booklet is available at [www.kidney.org.au](http://www.kidney.org.au)



## Do you want to know more?



Kidney Health Australia provides accredited education for health professionals through our Kidney Check Australia Taskforce (KCAT) program.

Accredited (RACGP, ACRRM, RCNA) face to face and online learning modules are available free of charge to Australian health professionals.

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

If you would like to complete some education related to the contents of this booklet, please visit [www.KCAT.org.au](http://www.KCAT.org.au) for further information.

## Resources for your practice

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Kidney Health Australia has a suite of brochures, health fact sheets and books that give precise, up to date health promotion and disease prevention messages. Copies can be obtained by downloading or ordering from [www.kidney.org.au](http://www.kidney.org.au), or by calling 1800 4 KIDNEY (543 639).

## Contact us

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The Kidney Health Information Service (KHIS) is a valuable and much demanded free call telephone service offering information, support, referral and consumer advice, which ensures people are directed to the most appropriate service to meet their needs. This service is suitable for the general Australian public, those living with or affected by kidney disease and allied health professionals. We invite people to utilise this service – please contact 1800 4 KIDNEY (543 639) or email your query to [KHIS@kidney.org.au](mailto:KHIS@kidney.org.au)

## Foreword

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Chronic kidney disease (CKD) is a major public health problem in Australia and throughout the world. It is estimated that 10% of all adults presenting to a general practice in Australia have CKD, and 80% have at least one risk factor for CKD.

Early CKD is usually asymptomatic and must be actively sought to be recognised. The main aim is to identify CKD in all high-risk adults and to manage it appropriately.

CKD is often present in the context of cardiovascular disease (CVD) and/or diabetes, and significantly increases the mortality and morbidity associated with these conditions. Fortunately, the optimal management of CKD overlaps with the best care for CVD and diabetes, leading to synchronous therapeutic strategies.

Most CKD identification is best performed in primary care and managed without referral to a specialist. Many aspects of CKD management lend themselves to involvement of a team approach, with the practice nurse potentially playing a pivotal role. In the absence of breakthrough cures, there is no other way the spiralling numbers of patients with kidney failure will be reduced.

The aim of this 2nd edition of the CKD Management in General Practice booklet is to provide guidance and clinical tips to help identify, manage and refer CKD in your practice. The recommendations contained in this booklet were formed from existing evidence-based clinical guidelines, current research, and clinical consensus. We hope that general practitioners and health professionals will consult these guidelines in order to ensure a high standard of care for their patients.

The front and back cover of the booklet highlights key points, and you may wish to remove this for easy access.



Associate Professor Tim Mathew  
National Medical Director



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## What's new?

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### **Blood pressure targets** (See Page 25 for more detail)

- People with CKD should be treated with blood-pressure lowering drugs to maintain a blood pressure that is consistently below 140/90 mmHg.
- If albuminuria is present (urine ACR  $>3.5$  mg/mmol in females and  $>2.5$  mg/mmol in males) a consistent blood pressure below 130/80 mmHg should be achieved.
- All people with diabetes should maintain a consistent blood pressure below 130/80 mmHg.

### **CKD Staging** (See Page 16 for more detail)

- It is now recommended by Australian and international guidelines that the stages of CKD should be based on the combined indices of kidney function (measured or estimated GFR), kidney damage (albuminuria/proteinuria), and underlying diagnosis (e.g., Stage 2 CKD with microalbuminuria secondary to diabetic kidney disease).
- Stage 3 CKD (eGFR 30-59 mL/min/1.73m<sup>2</sup>) has been divided into Stage 3a (eGFR 45-59 mL/min/1.73m<sup>2</sup>) and Stage 3b (eGFR 30-44 mL/min/1.73m<sup>2</sup>) to more accurately reflect risk stratification.
- eGFR is now calculated using the CKD-EPI formula as it improves risk stratification. This will make limited or no difference to your practice.

### **Testing for Albuminuria** (See Page 11 for more detail)

- The 2012 Australasian Proteinuria Consensus Working Group recommends that the preferred method for the detection of albuminuria in people with and without diabetes is urinary albumin:creatinine ratio (ACR).
- Urine ACR accurately predicts renal and cardiovascular risks in population studies, and reduction in urine ACR predicts renoprotective benefit in intervention trials.
- Dipstick for protein in the urine is now no longer recommended for this purpose as their sensitivity and specificity is not optimal.

### **eGFR and Elderly People** (See Page 14 for more details)

- The 2012 Creatinine Consensus Working Group recommends against the use of age-related decision points in adults.
- It is now known that an eGFR  $< 60$  mL/min/1.73 m<sup>2</sup> is very 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.



## What is Chronic Kidney Disease (CKD)?

### Chronic kidney disease (CKD) is diagnosed as:

- an estimated or measured 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 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)

### 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.

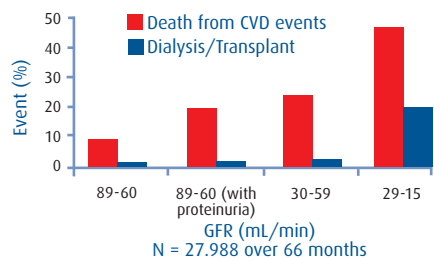


## Why worry about CKD?

**CKD is a significant and growing public health problem responsible for substantial burden of illness and premature mortality. In Australia, CKD is:**

- **Common**
  - 1 in 3 adults is at increased risk of developing CKD
  - 1 in 9 adults has some sign of CKD
  - 10% of people attending general practice have CKD, but most do not know it
- **Harmful**
  - Individuals with CKD have a 2 to 3-fold greater risk of cardiac death than individuals without CKD
  - Kidney and urinary tract diseases are the 9th leading cause of death in Australia, killing more people each year than breast cancer, prostate cancer and road deaths
  - Every day, 6 Australians commence expensive dialysis or transplantation to stay alive
- **Treatable**
  - 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

### Outcomes in patients with CKD

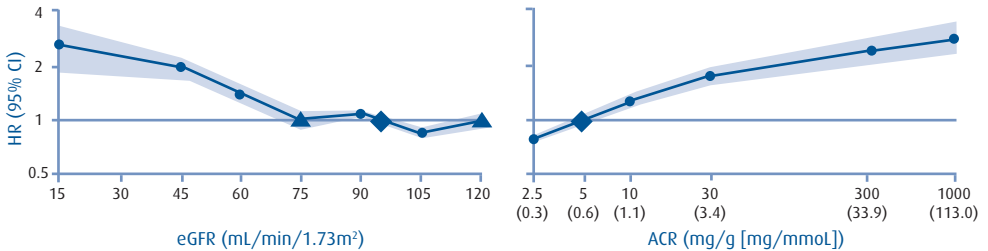


Source: Keith et al. Arch Intern Med 2004;164:659-63.

## CKD and cardiovascular disease

- Both reduced eGFR and significant albuminuria are independent risk factors for cardiovascular disease (CVD).
- Recent studies have confirmed that even early CKD constitutes a significant risk factor for cardiovascular events and death.
- For people with CKD, the risk of dying from cardiovascular events is up to 20 times greater than requiring dialysis or transplantation.

### Hazard ratios & 95% CIs for cardiovascular mortality according to eGFR & urine ACR



Source: Chronic Kidney Disease Prognosis Consortium, Lancet 2010; 375(9731):2073-81

## Absolute Cardiovascular Risk Assessment

- A comprehensive risk assessment, using an absolute risk approach, is recommended to assist general practitioners effectively manage their patient's CVD risk by providing a meaningful and individualised risk level.
- Absolute risk is the numerical probability of an event occurring within a specified period, expressed as a percentage. For example, if your patient's risk is 15%, there is a 15% probability that they will experience a cardiovascular event within 5 years.
- Absolute cardiovascular risk assessment using the Australian CVD tool to predict the risk of a cardiovascular event over the next 5 years should be performed for all adults aged 45-74 years without existing CVD and without a clinically determined risk factor.
- People with moderate or severe CKD, defined as persistently having a urine ACR >30 mg/mmol (equivalent to dipstick 1+ proteinuria) or eGFR <45 mL/min/1.73m<sup>2</sup>, are considered to be at the highest risk of a cardiovascular event and therefore should not be assessed by the absolute risk tool.
- For these people, identifying all cardiovascular risk factors present will enable intensive management by lifestyle interventions (for all patients) and pharmacological interventions (where indicated).





## Who is at risk of CKD?

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- 1 in 3 adult Australians is at an increased risk of developing CKD.
- Adult Australians are at increased risk of developing CKD if they:
  - are 60 years or older
  - have diabetes
  - have a family history of kidney disease
  - have established cardiovascular disease
  - have high blood pressure
  - are obese (body mass index  $\geq 30$ )
  - are a smoker
  - are of Aboriginal or Torres Strait Islander origin\*

\*The greater prevalence of CKD in some Aboriginal and Torres Strait Islander communities is due to the high incidence of the risk factors outlined above, in addition to increased levels of inadequate nutrition, alcohol abuse, streptococcal throat and skin infection, and poor living conditions.

## What are the causes of kidney disease?

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**The most common causes of end stage kidney disease (ESKD) in Australia are:**

- diabetic nephropathy
- glomerulonephritis
- hypertensive vascular disease
- reflux nephropathy

**When CKD is initially diagnosed it is important to consider the underlying cause and to pursue the diagnosis sufficiently to exclude conditions with specific treatments, such as:**

- obstruction
- vasculitis
- nephritic syndrome
- rapidly progressing glomerulonephritis

Irrespective of the underlying cause of CKD the treatment of complications follows the principles outlined in this booklet.



## Detection of CKD

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### Importance of Early Detection

- Increasing amounts of albumin in the urine correlate directly with an increased rate of progression to end-stage kidney disease.
- The amount of albuminuria in the urine can be reduced significantly by the use of an ACE inhibitor or ARB.
- Reduction in the amount of albuminuria is associated with improved outcomes.
- Early intervention in CKD can reduce progression and cardiovascular risk by up to 50%, and may also improve quality of life.

## Clinical Presentation of CKD

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### 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 ESKD requiring dialysis or transplant (eGFR  $<15$  mL/min/1.73m<sup>2</sup>).

### Symptoms of ESKD include:

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



## Initial testing for CKD

- 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.
- 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**.
- Serum creatinine is an insensitive marker for detecting mild to moderate kidney disease – eGFR is the preferred test.
- 50% or more of kidney function can be lost before the serum creatinine rises above the upper limit of normal.

## Early detection of CKD using Kidney Health Check

Indication for testing <sup>*</sup>	Recommended tests	Frequency of testing
Smoker	Urine ACR <sup>§</sup> , eGFR, blood pressure	Every 1 - 2 years <sup>**</sup>
Diabetes		
Hypertension		
Obesity		
Established cardiovascular disease <sup>†</sup>		
Family history of CKD		
Aboriginal or Torres Strait Islander origin aged ≥ 30 years <sup>‡</sup>		

Source: RACGP Red Book 8th edition (2012, in press) and National Guide to a preventive health assessment in Aboriginal and Torres Strait Islander peoples (NACCHO) (2012, in press).

\* 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 test these individuals for kidney disease.

\*\* 1 year for individuals with hypertension or diabetes

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

‡ See Page 10 for more detail regarding indication for testing in Aboriginal and Torres Strait Islander peoples.

§ If urine ACR positive arrange 2 further tests over 3 months (preferably first morning void).  
If eGFR < 60mL/min/1.73m<sup>2</sup> repeat within 14 days.

## Aboriginal and Torres Strait Islander peoples

- Age-standardised incidence of ESKD is significantly higher in Aboriginal and Torres Strait Islander peoples compared with non Aboriginal and Torres Strait Islander peoples.

### Recommendations for CKD detection in Aboriginal and Torres Strait Islander peoples

Indication for testing	Recommended tests	Frequency of testing
People 18-29 years without any CKD risk factors	Assess for CKD risk factors (overweight and obesity, diabetes, elevated blood pressure, smoking, and family history of kidney disease)	As part of annual health assessment
People 18-29 years with one of the following CKD risk factors: <ul style="list-style-type: none"> <li>• Family history of CKD or premature CVD</li> <li>• Overweight/obesity</li> <li>• Smoking</li> <li>• Diabetes</li> <li>• Elevated blood pressure</li> </ul> All people ≥30 years	Urine ACR, eGFR, blood pressure	Every two years (or more frequently if CVD risk is elevated)

Note. If urine ACR positive arrange 2 further tests over 3 months (preferably first morning void).  
 If eGFR < 60mL/min/1.73m<sup>2</sup> repeat within 14 days.

Source: National Guide to a preventive health assessment in Aboriginal and Torres Strait Islander peoples (NACCHO) (2012, in press).

### Benefits of identifying Aboriginal and Torres Strait Islander peoples:

- Clinician awareness of increased risk of CKD and cardiovascular disease and importance of screening other family members for CKD.
- Individuals able to access annual health check (Medicare item 715).
- Individuals eligible for Aboriginal and Torres Strait Islander peoples-specific pharmaceutical benefits.
- Individuals are eligible for “Close the Gap” PBS co-payments.
- The Aboriginal and Torres Strait Islander community becomes engaged with the health care system.

For further detailed information refer to the NACCHO National Guide to a preventive health assessment in Aboriginal and Torres Strait Islander peoples ([www.naccho.org.au](http://www.naccho.org.au))



## Tests used to investigate CKD

### Urine tests - Albuminuria

- 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 very rare for an individual to have increased excretion of non-albumin proteins without concomitant increased excretion of albumin.

#### Clinical Tip

The preferred method for assessment of albuminuria in both diabetes and non-diabetes is urinary ACR measurement in a first void spot specimen. Where a first void specimen is not possible or practical, a random spot urine specimen for urine ACR is acceptable.



### How to detect albuminuria\*

- Urine ACR accurately predicts renal and cardiovascular risks in population studies, and reduction in urine ACR predicts renoprotective benefit in intervention trials.
- Urine ACR exhibits greater sensitivity than protein:creatinine ratio (PCR) for detecting lower amounts of clinically important albuminuria.
- 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 (including the initial test). CKD is present if the albuminuria is persistent for at least three months.
- If the first positive ACR is a random spot (as it may be for opportunistic testing), then repeat tests should ideally be first morning void specimens.
- Dipstick for protein in the urine is now no longer recommended for this purpose as the sensitivity and specificity is not optimal.
- Urine PCR can be used for quantification and monitoring of proteinuria if required, but this is not preferred.

### Approx. equivalents between urine ACR and other measures of protein excretion

	Urine ACR (mg/mmol)	24h urine albumin (mg/day)	Urine PCR (mg/mmol)	24h urine protein (mg/day)
<b>Microalbuminuria</b>	Male: 2.5-25 Female: 3.5-35	30-300	Male: 4-40 Female: 6-60	50-500
<b>Macroalbuminuria</b>	Male: > 25 Female: > 35	> 300	Male: > 40 Female: > 60	> 500

\* Source: Australasian Proteinuria Consensus Working Group (2012).

## Factors Other than CKD known to Increase Urine Albumin Excretion

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

### 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.
- 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.

#### Clinical Tip



Under the age of 40, isolated haematuria (haematuria without albuminuria, reduced GFR, or urinary tract malignancy) is usually consistent with a mild underlying glomerulonephritis with a low propensity for progression.

### How to detect haematuria\*

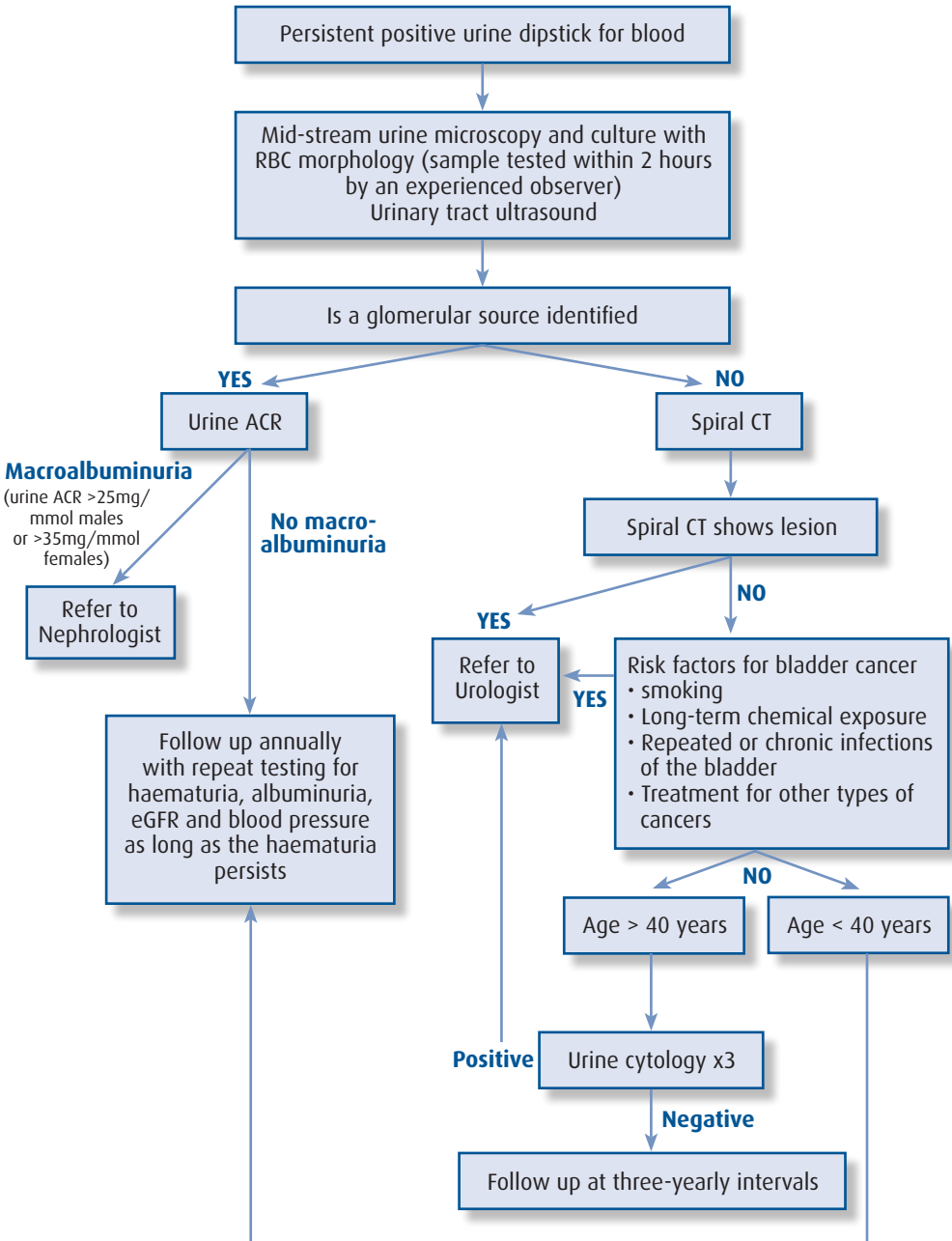
- Use dipsticks rather than urine microscopy as they are more sensitive.
- Evaluate further if there is a result of 1+ or more.

Persistent microscopic (invisible) haematuria	Action
To differentiate, in the absence of albuminuria, from transient haematuria	Confirm persistent microscopic haematuria by 2 out of 3 positive dipstick tests
With or without albuminuria	Investigate for urinary tract malignancy in appropriate age groups
Without albuminuria and urinary tract malignancy excluded (presumed glomerular bleeding)	Follow up annually with repeat testing for haematuria, albuminuria, eGFR and blood pressure monitoring as long as the haematuria persists Screen family members for haematuria

\* Source: Halpin D, Stevens P, Bakhshi L et al (2008).



# Algorithm for management of persistent microscopic haematuria



## Blood tests – glomerular filtration rate (GFR)\*

- GFR is accepted as the best measure of kidney function.
- GFR can be estimated (eGFR) from serum creatinine using prediction equations.
- eGFR is recommended to be automatically reported (using the CKD-EPI equation) with every request for serum creatinine in individuals aged  $\geq 18$  years.
- eGFR values may be reported as precise figures up to at least  $90 \text{ mL/min/1.73 m}^2$ . Values greater than this may be reported as a precise figure or as  $> 90 \text{ mL/min/1.73 m}^2$  depending on laboratory preference.
- Further investigation of reduced eGFR is only required if the eGFR is  $< 60 \text{ mL/min/1.73 m}^2$ .
- If eGFR is  $< 60 \text{ mL/min/1.73 m}^2$ , consider clinical situations where eGFR results may be unreliable and/or misleading and retest within 14 days.
- If an initial eGFR measurement is  $< 60 \text{ mL/min/1.73 m}^2$ , it is suggested that subsequent measurements of serum creatinine and calculation of eGFR are carried out when the individual has fasted or specifically avoided a cooked meat meal in the four hours prior to blood sampling.
- An eGFR  $< 60 \text{ mL/min/1.73 m}^2$  is common in older people, but is predictive of significantly increased risks of adverse clinical outcomes, and should not be considered physiological or age-appropriate.

### Clinical Tip

A measured or estimated GFR  $< 60 \text{ mL/min/1.73 m}^2$  is associated with increased risks of adverse renal, cardiovascular and other clinical outcomes, irrespective of age.



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

- acute changes in kidney function (e.g. acute kidney failure)
- people on dialysis
- 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 overestimate eGFR) or high muscle mass (may underestimate eGFR)
- children under the age of 18 years
- severe liver disease present
- eGFR values above  $90 \text{ mL/min/1.73 m}^2$
- drugs interacting with creatinine excretion (e.g. fenofibrate, trimethoprim)

\* Source: Australasian Creatinine Consensus Working Group (2012).





## Use of eGFR in various ethnic populations

- The CKD-EPI formula is a useful tool to estimate GFR in all people, including various ethnic populations.
- The CKD-EPI formula has been validated as a tool to estimate GFR in some non-Caucasian populations, including South-East Asian, African, Indian and Chinese individuals living in Western countries.
- The different methods to estimate GFR from serum creatinine have not been validated in people of Aboriginal or Torres Strait Islander origin.
- Validation studies are underway, and until these are reported it is considered advisable to continue using eGFR in people of Aboriginal or Torres Strait Islander origin.

## eGFR and drug dosing

- Dose reduction of some drugs is recommended for people with reduced kidney function (See Page 19).
- Both eGFR (mL/min/1.73m<sup>2</sup>) and estimated CrCl (mL/min) provide an estimate of renal drug clearance.
- If using eGFR for drug dosing body size should be considered, in addition to referring to the approved Product Information.
- For drugs with a narrow therapeutic index, therapeutic drug monitoring or a valid marker of drug effect should be used to individualise dosing.
- For drug dosing in very large or very small people, it may be preferred to calculate an eGFR that is not normalised to 1.73m<sup>2</sup> body surface area (BSA).
- To revert to an uncorrected eGFR, the eGFR result from the CKD-EPI should be multiplied by the individual's BSA divided by 1.73 to generate an eGFR in mL/min.

## 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.

## Further evaluation

- If the abnormal eGFR is confirmed on repeat testing, further assessment is recommended:
- Urine ACR (preferably on first morning void, although a random urine is acceptable)
- Confirmatory serum urea/electrolytes/creatinine
- Blood pressure
- Full blood count
- Fasting lipids and glucose
- Urine culture and specificity, including dipstick for haematuria
- Urinary tract ultrasound scan

Depending on the age of the individual and the severity of CKD, consideration may also be given to iron studies, serum calcium, phosphate and parathyroid hormone.

## Staging of CKD\*

- Combine Kidney Function Stage (stage 1-5) with description of kidney damage (albuminuria) and clinical diagnosis to specify CKD fully (e.g., Stage 2 CKD with microalbuminuria, secondary to diabetic kidney disease).



### Clinical Tip

For people with CKD, the combination of low GFR and albuminuria places them at greater risk of CKD and CVD progression at all ages than those with just one of low GFR or albuminuria.

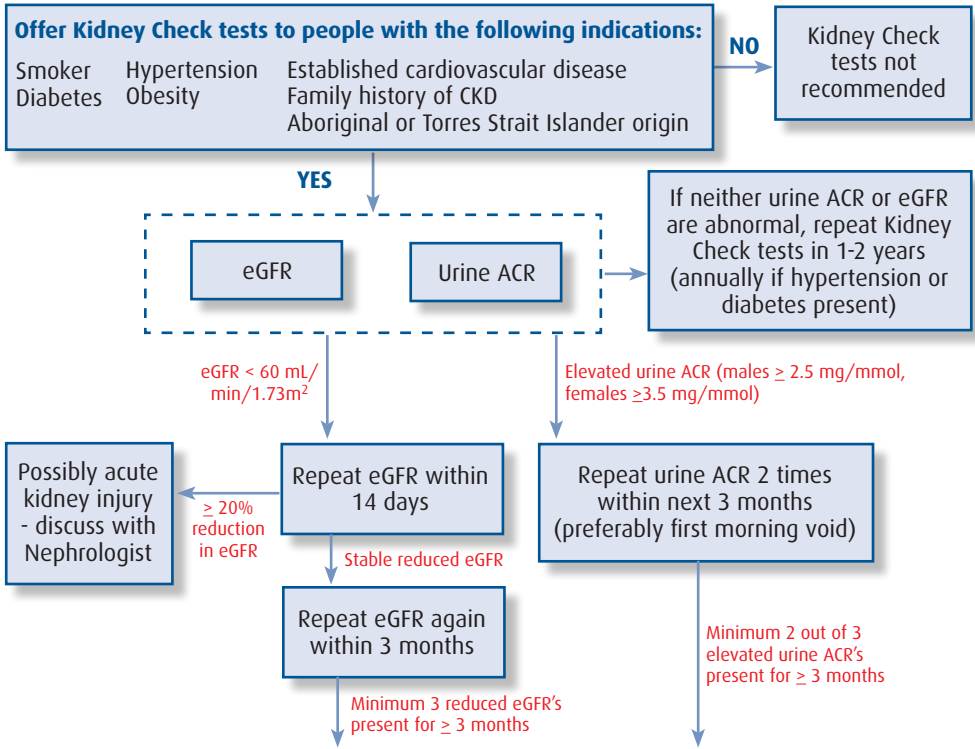
Kidney Function Stage	GFR (mL/min/1.73m <sup>2</sup> )	Albuminuria Stage		
		Normal (urine ACR mg/mmol) Male: < 2.5 Female: < 3.5	Microalbuminuria (urine ACR mg/mmol) Male: 2.5-25 Female: 3.5-35	Macroalbuminuria (urine ACR mg/mmol) Male: > 25 Female: > 35
1	≥90	Not CKD unless haematuria, structural or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

Refer to colour-coded Clinical Action Plans for management strategies

\* Source: KHA-CARI Guidelines. Early chronic kidney disease: diagnosis, classification and staging of chronic kidney disease (2012).



# Algorithm for initial detection of CKD



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

Combine eGFR stage (1-5), albuminuria stage and underlying diagnosis to fully specify CKD stage (eg., stage 2 CKD with microalbuminuria secondary to diabetic kidney disease)  
Refer to colour-coded action plans for management strategies

## Indications for referral to a Nephrologist\*

### Appropriate referral is associated with:

- reduced rates of progression to ESKD
- decreased patient morbidity and mortality
- decreased need for and duration of hospitalisation
- increased likelihood of permanent dialysis access created prior to dialysis onset
- increased likelihood of kidney transplantation
- timely predialysis education

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

- eGFR  $< 30$  mL/min/1.73m<sup>2</sup>
- Persistent significant albuminuria (urine ACR  $\geq 30$  mg/mmol)
- A consistent decline in eGFR from a baseline of  $< 60$  mL/min/1.73m<sup>2</sup> (a decline  $> 5$  mL/min/1.73m<sup>2</sup> over a six-month period which is confirmed on at least three separate readings)
- Glomerular haematuria with macroalbuminuria
- CKD and hypertension that is hard to get to target despite at least three anti-hypertensive agents

Also take into account the individual's wishes and comorbidities when considering referral.

**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 necessary if:

- Stable eGFR  $\geq 30$  mL/min/1.73m<sup>2</sup>
- Urine ACR  $< 30$  mg/mmol (with no haematuria)
- Controlled blood pressure

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

### Tips for referral:

- Don't refer to Nephrologist if targets of therapy are achieved.
- Pay attention to CVD risk reduction.
- Ensure the individual has had a recent urine ACR, current blood chemistry and haematology, and a urinary tract ultrasound scan.
- Spiral CT angiogram for hypertension is not recommended without specialty advice.
- Consider discussing management issues with a Nephrologist in cases where uncertainty regarding referral exists.

\* Source: KHA-CARI Guidelines. Early chronic kidney disease: When to refer for specialist renal care (2012).



## Medications in CKD

- Dosage reduction or cessation of renally excreted medications is generally required once the GFR falls below 60 mL/min/1.73m<sup>2</sup>.
- It is important to review renally excreted medications, as well as avoid nephrotoxic medications in people with CKD.

### Commonly prescribed drugs that may need to be reduced in dose or ceased in CKD:

- Antivirals
- Benzodiazepines
- Colchicine
- Dabigatran
- Digoxin
- Fenofibrate
- Gabapentin
- Glibenclamide
- Insulin
- Lithium
- Metformin (use with caution if GFR 30-60 mL/min/1.73m<sup>2</sup>; not recommended if GFR < 30 mL/min/1.73m<sup>2</sup>)
- Opioid analgesics
- Sotalol
- Spironolactone
- Valaciclovir

### Commonly prescribed drugs that can adversely affect kidney function in 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
- Calcineurin inhibitors



#### Clinical Tip

The combination of ACE inhibitor (or ARB), diuretic and NSAID (except low-dose aspirin) can result in a potentially serious interaction, 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.

### Effect of ACE inhibitors and ARBs on kidney function

- ACE inhibitors and ARBs can cause a reversible reduction in GFR when treatment is initiated.
- If the reduction is less than 25% and stabilises within two months of starting therapy, the ACE inhibitor or ARB should be continued.
- If the reduction in GFR exceeds 25% below the baseline value, the ACE inhibitor or ARB should be ceased and consideration given to referral to a Nephrologist for investigation of possible bilateral renal artery stenosis.
- Combined therapy of ACE inhibitor and ARB is not recommended except with specialist advice.
- Rises in serum K<sup>+</sup> of up to 0.5mmol/L are expected.
- ACE inhibitors and ARBs can safely be prescribed at all stages of CKD and should not be deliberately avoided just because GFR is reduced.

## Yellow Clinical Action Plan

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**eGFR  $\geq 60$  mL/min/1.73m<sup>2</sup> with microalbuminuria  
or eGFR 45-59 mL/min/1.73m<sup>2</sup> with normoalbuminuria**

### Goals of management

- investigations to exclude treatable kidney disease (see Page 11-15)
- assessment of absolute cardiovascular risk
- reduce CVD risk
- avoidance of nephrotoxic medications (see Page 19) or volume depletion

### Monitoring

- 12 monthly clinical review
- clinical assessment
  - blood pressure
  - weight
- laboratory assessment
  - urine ACR
  - biochemical profile including urea, creatinine and electrolytes
  - eGFR
  - HbA1c (for people with diabetes)
  - fasting lipids

### Absolute cardiovascular risk assessment

- The presence of CKD is one of the most potent known risk factors for CVD.
- Perform absolute cardiovascular risk assessment using the Australian CVD tool for all adults aged 45-74 years (35 years and above for Aboriginal and Torres Strait Islander peoples) without existing CVD and without a clinically determined risk factor (see NVDPA Guidelines (2009) for more detail).
- Provide lifestyle and pharmacological management strategies (if indicated) based on the patient's risk level and clinical judgement (e.g., high risk required more intensive intervention and follow up).

### Lifestyle modification

- Lifestyle modification: cessation of smoking, weight reduction, low-salt diet, physical activity, and moderate alcohol consumption are successful in reducing overall CVD risk.
- Refer to SNAP guide for management of lifestyle risk factors.

## Yellow Clinical Action Plan

**eGFR  $\geq 60$  mL/min/ $1.73\text{m}^2$  with microalbuminuria  
or eGFR 45-59 mL/min/ $1.73\text{m}^2$  with normoalbuminuria**

### Blood pressure reduction

- CKD can cause and aggravate hypertension, and hypertension can contribute to the progression of CKD.
- Reducing blood pressure to below threshold levels is one of the most important goals in management of CKD (see blood pressure targets on Page 25).
- ACE inhibitor or ARB is recommended as first line therapy.
- Combined therapy of ACE inhibitor and ARB is not recommended.
- Maximal tolerated doses of ACE inhibitor or ARB is recommended.
- Hypertension may be difficult to control and multiple (3 - 4) medications are frequently required.

### Clinical Tip



ACE inhibitors and ARBs can cause a reversible reduction in GFR when treatment is initiated. If the reduction is less than 25% and stabilises within two months of starting therapy, the ACE inhibitor or ARB should be continued. If the reduction in GFR exceeds 25% below the baseline value, the ACE inhibitor or ARB should be ceased and consideration given to referral to a Nephrologist for bilateral renal artery stenosis.

### Lipid-lowering treatments

- Lipid-lowering treatment should be considered where appropriate for CVD risk reduction.

### Glycaemic control

- For people with diabetes, blood glucose control significantly reduces the risk of developing CKD, and in those with CKD reduces the rate of progression.

## Orange Clinical Action Plan

**eGFR 30-59 mL/min/1.73m<sup>2</sup> with microalbuminuria  
or eGFR 30-44 mL/min/1.73m<sup>2</sup> with normoalbuminuria**

### Goals of management

- investigations to exclude treatable disease (see Page 11-15)
- reduce progression of kidney disease
- reduce CVD risk
- early detection and management of complications (see Page 25-33)
- avoidance of nephrotoxic medications (see Page 19) or volume depletion
- adjustment of medication doses to levels appropriate for kidney function (see Page 19)
- appropriate referral to a Nephrologist when indicated (see Page 18)

### Monitoring

- 3-6 monthly clinical review
- clinical assessment
  - blood pressure
  - weight
- laboratory assessment
  - urine ACR
  - biochemical profile including urea, creatinine and electrolytes
  - eGFR
  - HbA1c (for people with diabetes)
  - fasting lipids
  - full blood count
  - calcium and phosphate
  - parathyroid hormone (6-12 monthly if eGFR < 45 mL/min/1.73m<sup>2</sup>)

### Absolute cardiovascular risk assessment

- People with moderate or severe CKD (defined as persistently having a urine ACR >30 mg/mmol or eGFR <45mL/min/1.73 m<sup>2</sup> are considered to be at the highest risk of a cardiovascular event and do not need to be assessed by the cardiovascular risk tool.
- For these groups, identifying all cardiovascular risk factors present will enable intensive management by lifestyle interventions (for all patients) and pharmacological interventions (where indicated).

See also lifestyle modification, blood pressure reduction, lipid-lowering treatments, and glycaemic control as per Yellow Action Plan.





# Red Clinical Action Plan

## Macroalbuminuria irrespective of eGFR or eGFR <30 mL/min/1.73m<sup>2</sup> irrespective of albuminuria

### Goals of management

- appropriate referral to a Nephrologist when indicated (see Page 18)
- prepare for dialysis or pre-emptive transplant if eGFR <30 mL/min/1.73m<sup>2</sup>
- discuss advanced care directive if dialysis inappropriate
- reduce progression of kidney disease
- reduce CVD risk
- early detection and management of complications (see Page 25-33)
- avoidance of nephrotoxic medications (see Page 19)
- adjustment of medication doses to levels appropriate for kidney function (see Page 19)
- multidisciplinary team involvement (see Page 34)

### Monitoring

- 1-3 monthly clinical review
- clinical assessment
  - blood pressure
  - weight
  - oedema
- laboratory assessment
  - urine ACR
  - biochemical profile including urea, creatinine and electrolytes
  - eGFR
  - HbA1c (for people with diabetes)
  - fasting lipids
  - full blood count (if anaemic see Page 30)
  - calcium and phosphate
  - parathyroid hormone (6-12 monthly if eGFR < 45 mL/min/1.73m<sup>2</sup>)
  - advanced care planning

### Absolute cardiovascular risk assessment

- People with moderate or severe CKD (defined as persistently having a urine ACR >30 mg/mmol or eGFR <45mL/min/1.73 m<sup>2</sup>) are considered to be at the highest risk of a cardiovascular event and do not need to be assessed by the cardiovascular risk tool.
- For these groups, identifying all cardiovascular risk factors present will enable intensive management by lifestyle interventions (for all patients) and pharmacological interventions (where indicated).



## Referral to a Nephrologist

The KHA-CARI guidelines recommend that individuals 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 m<sup>2</sup>).

See Page 18 for referral guidelines.

While renal services undertake much of the CKD management during this stage, it is important that the individual maintains contact with their regular General Practitioner to ensure coordination of whole-patient care, routine screening and health promotion, and psychosocial support.

## Pre-emptive Transplantation

Pre-emptive transplantation means receiving a kidney transplant from a live donor prior to initiation of dialysis. Pre-emptive transplantation is associated with:

- reduced risk of death
- longevity 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/1.73m<sup>2</sup>), but before dialysis is needed.

## 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 non-dialysis treatment (no dialysis or transplantation), and palliative care arrangements.

See also lifestyle modification, blood pressure reduction, lipid-lowering treatments, and glycaemic control as per Yellow Action Plan.



## Common CKD complications

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

### Hypertension

Target:  $\leq 140/90$  mmHg, or  $\leq 130/80$  mmHg in people with albuminuria (urine ACR  $>3.5$  mg/mmol in females and  $>2.5$  mg/mmol in males)

Source: KDIGO (2012).

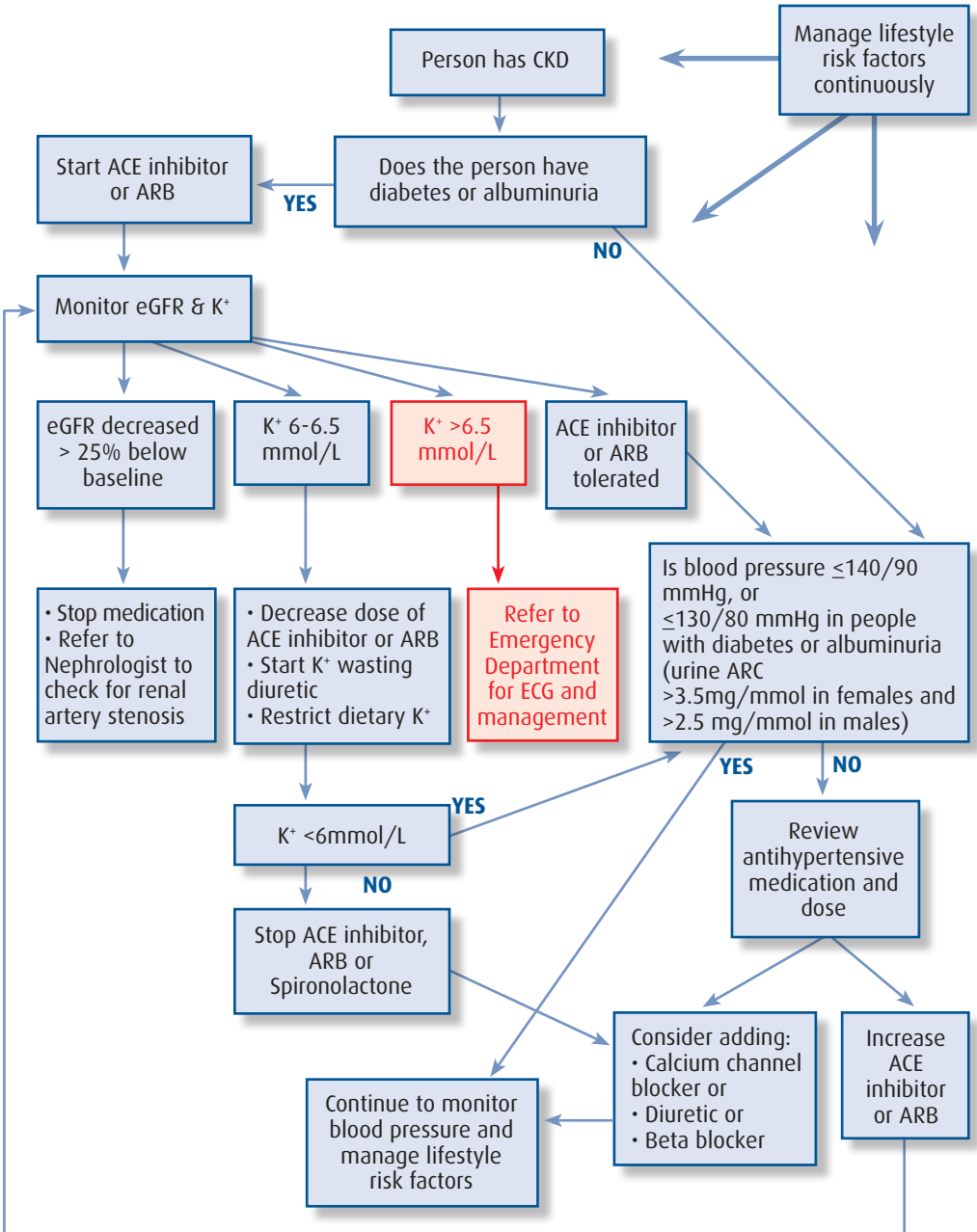
Hypertension is both a cause of CKD and a complication of CKD and can be difficult to control. The risks of uncontrolled hypertension include progression of kidney disease and increased risk of coronary heart disease and stroke.

### Management

- Lifestyle – refer to SNAP guidelines.
- Multiple medications (often 3 or more drugs) will be needed to control hypertension adequately in most people with CKD.
- Consider sleep apnoea as a cause of resistant hypertension in the obese
- People with diabetes or proteinuria should be treated with an ACE inhibitor or ARB as first line therapy.
- When treatment with an ACE inhibitor or ARB is initiated, the GFR can decrease and potassium levels can rise.
  - If the acute decrease in GFR is less than 25% below the baseline level and stabilises within two months, the medication should be continued. People whose GFR decreases are most likely to achieve the greatest benefit in terms of kidney protection.
  - If the decrease in GFR is greater than 25% below 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 be stopped.
- Diuretics should be used in most individuals. Both non loop 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.
- Beta-blockers may be useful in people with coronary heart disease, tachyarrhythmias and heart failure, but are contraindicated in asthma and heart block.
- Calcium channel blockers may be used for people with angina, the elderly and those with systolic hypertension.
- Combined therapy of ACE inhibitor and ARB is not recommended.



# Algorithm for management of hypertension in people with CKD



**Albuminuria****Target:**

50% reduction in urine ACR

Source: KHA-CARI (2012).

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

**Management**

- ACE inhibitor or ARB as first-line therapy.
- Reduction in salt output through reducing oral salt intake.
- Spironolactone (use with caution).

**Lipids****Target:**

Total <4.0 mmol/L  
LDL <2.5 mmol/L

Source: National Heart Foundation (2005).

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 proteinuria, particularly those with nephrotic syndrome. Dyslipidaemia should be treated as per CVD recommendations and targets.

**Management**

- Dietary advice.
- Statins (dose reduction not necessary).

**Glycaemic Control****Target:**

Pre-prandial BSL  
4.0-6.0 mmol/L  
HbA1c <7.0%

Source: Diabetes Australia (2011).

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.

**Management**

- Lifestyle modification.
- Oral hypoglycaemics.
- Insulin.
- Gliptins.
- Incretin mimetics.

### Mineral and Bone Disorder

#### Target:

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

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

PTH 2-9 x upper limit of normal and avoid trends towards the extremes of this range

Vitamin D (25-hydroxyvitamin D) optimal levels may be > 75 nmol/L

Source: KDIGO (2006), KHA-CARI (2012), K/DOQI (2003).

Changes in the metabolism of calcium, phosphate, parathyroid hormone and Vitamin D typically start to occur once  $GFR \leq 60 \text{ mL/min/1.73m}^2$ .

As kidney function decreases, the renal clearance of phosphate is diminished, leading to higher serum phosphate levels and levels of calcitriol fall. Calcitriol is the most active form of vitamin D and requires kidney function for its synthesis. Calcium levels may fall as a result of less vitamin D dependent calcium uptake from the gastrointestinal tract.

The combined effects of higher phosphate, lower calcium and lower vitamin D levels all serve to stimulate parathyroid hormone production, and in turn elevated levels of PTH increase the resorption and release of mineral from bone.

These changes are associated with an increased risk of fracture and also increased cardiovascular mortality, perhaps mediated by accelerated vascular calcification.

## Management

- Phosphate
  - Dietary restriction of phosphate. This is best managed with advice from a renal dietitian.
  - Use of phosphate binders, which bind dietary phosphate to prevent absorption. Commonly used binders are typically calcium-based.
  - Sevelamer or lanthanum are also available for individuals on dialysis.
- Calcium
  - If phosphate is controlled, calcium will typically remain in normal range. If the level is low with normal phosphate level consider Vitamin D supplementation.
- Vitamin D
  - Cholecalciferol, the form of vitamin D that comes from sun exposure, can be given as a dietary supplement and will be converted to 25-hydroxyvitamin D by the liver.
  - If kidney function is still intact, it will then be converted to calcitriol, the most active form and will help to suppress the development of secondary hyperparathyroidism.
  - Calcitriol, the most active form of vitamin D is also used in CKD for suppression of secondary hyperparathyroidism and is the preferred vitamin D in later stages of CKD when kidney function is very poor.
  - Calcitriol is available on PBS Authority for "the indication of hypocalcaemia due to renal disease". The major side effect of therapy with calcitriol is hypercalcaemia and hyperphosphatemia.



- Parathyroid hormone
  - The 'calcimimetic' cinacalcet can be used. This drug binds to a receptor for calcium on the parathyroid gland and can be used to suppress production of parathyroid hormone. Treatment with cinacalcet tablets generally leads to an improvement in levels of PTH and phosphate and a lowering of calcium levels. Cinacalcet is currently only available for individuals on dialysis.
  - In people with CKD and severe hyperparathyroidism who fail to respond to medical/pharmacological therapy, parathyroidectomy should be considered, particularly when calcium or phosphate levels cannot be satisfactorily controlled.

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

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

**Anaemia\***

**Target:** Hb 100 – 115 g/L  
Prior to commencement of epoetin: Ferritin > 100 µg/L  
TSAT >20%  
Once epoetin commenced:  
Ferritin 200-500 µg/L  
TSAT 30-40%

Anaemia of CKD is related to both:

- reduced erythropoietin production by the kidney
- resistance to the action of erythropoietin

Anaemia related to CKD usually occurs at GFRs of <60 mL/min/1.73m<sup>2</sup>.

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 any other cause should be excluded (e.g., blood loss).
  - In people with CKD treated with erythropoiesis stimulating agents (ESA or EPO) iron supplementation is typically required. This can be given either as oral iron or not infrequently as intravenous supplementation. Intramuscular iron is not recommended.
- 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 is available on the PBS for people with CKD and Hb < 100g/L.
- Treatment 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.
- Target Hb levels are lower in those individuals with past cerebrovascular accident or cancer, where the lowest dose of ESA is used to prevent blood transfusion.
- 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 and iron stores monthly. The aim is a rise of Hb at a rate of approximately 10g/L/month. Rapid correction of anaemia has been associated with hypertension and seizures.
  - Maintenance - target Hb is not fully defined in CKD, but the range is between 100-115 g/L. There is evidence of potential harm when Hb is targeted to exceed 130 g/L. Monitoring of Hb and iron studies is generally at three monthly intervals during this phase.

\*Source: KHA-CARI (2011), K/DOQI (2006).



**Dietary protein****Target:**

No lower than  
0.75 g/kg body  
weight/day

Source: KHA-CARI (2012).

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.

**Management**

- Dietary advice.

**Malnutrition****Target:**

Serum albumin  
≥35 g/L

Source: KHA-CARI (2012).

Poor food intake due to the symptoms of CKD can lead to malnutrition and low serum albumin.

**Management**

- Dietary advice.

**Uraemia**

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 are the substances we measure, the symptoms are most likely due to the accumulation of other toxic end products. These symptoms can lead to poor food intake and malnutrition. By the time uraemia becomes symptomatic dialysis is typically indicated.

**Management**

- Dialysis should be commenced as soon as uraemic symptoms develop
- If dialysis is not planned:
  - A low protein diet will help control gastrointestinal symptoms
  - Fluid control should be strict to avoid pulmonary oedema
  - Avoid unnecessary medications
  - Anti-emetics are of limited value

**Acidosis**

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

**Management**

- Supplementation with sodium bicarbonate (SodiBic) capsules may be considered in people with acidosis.
- Increased sodium load may worsen blood pressure control.

**Hyperkalaemia****Target:** $K^+ \leq 6.0$  mmol/L

Source: KHA-CARI (2005).

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 and weakness with reduced reflexes.

**Management**

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

**Restless Legs**

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

**Management**

- Iron replacement therapy.
- Home therapies such as massage, warm baths, warm/cool compresses, relaxation techniques, exercise.
- Dopaminergic agents or dopamine agonists (e.g. Levodopa, Ropinirole).
- Benzodiazepines.

**Sleep Apnoea**

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

**Management**

- Weight reduction.
- Avoid central nervous system depressants.
- CPAP therapy (if obstructive pattern)



## 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

- Assess routinely for depression.
- Implement psychosocial interventions such as cognitive behavioural therapy, structured problem solving, interpersonal therapy and social work support.
- 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.

## Nutrition

- People with CKD should be encouraged to eat a balanced and adequate diet according to energy requirements in line with the Dietary Guidelines of Australian Adults recommended by NMHRC.
- KHA-CARI guidelines recommend that people with progressive CKD should have individualised diet intervention involving an Accredited Practising Dietitian.
- Overweight or obese people with CKD should be prescribed caloric restriction under the management of an Accredited Practising Dietitian.

### Nutrition targets for people with early CKD (eGFR $\geq$ 30mL/min/1.73m<sup>2</sup>)

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

Source: KHA-CARI Guidelines. Modification of lifestyle and nutrition interventions for management of early chronic kidney disease (2012).



## Multidisciplinary care

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

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

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

- Supporting and assisting the individual 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 individual progresses to ESKD and has regular contact with the dialysis or transplant team, the General Practitioner, practice nurse, practice staff and other health professionals remain vital to optimal care.

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



## References

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- ANZDATA. The 32nd ANZDATA Registry Report. Australia and New Zealand Dialysis and Transplant Registry, Adelaide, South Australia; 2010.
- Atkins RC, Briganti EM, Zimmet PZ, Chadban SJ. Association between albuminuria and proteinuria in the general population: the AusDiab Study. *Neph Dial Transplant* 2003;18:2170-4.
- Australasian Proteinuria Consensus Working Group. Chronic kidney disease and measurement of albuminuria/proteinuria: A Position Statement. *Med J Aust* 2012; in press.
- Australasian Creatinine Consensus Working Group. Chronic kidney disease and automatic reporting of estimated glomerular filtration rate: New developments and revised recommendations. *Med J Aust* 2012; in press.
- Australasian Creatinine Consensus Working Group. Chronic kidney disease and automatic reporting of estimated glomerular filtration rate: a position statement. *Med J Aust* 2005;183(3):138-41.
- Australian Government Department of Health and Ageing, National Health and Medical Research Council. *Australian Immunisation Handbook*. 9th edition ed. Australia; 2007.
- Cervelli M. *The Renal Drug Reference Guide*. Kidney Health Australia, Melbourne, Victoria; 2007.
- Chadban SJ, Briganti EM, Kerr PG et al. Prevalence of kidney damage in Australian adults: The AusDiab kidney study. *J Am Soc Nephrol* 2003 July;14(7 Suppl 2):S131-S138.
- Chronic Kidney Disease Prognosis Consortium. Association of estimated glomerular filtration rate and albuminuria with all-cause and cardiovascular mortality in general population cohorts: a collaborative meta-analysis. *The Lancet* 2010;375(9731):2073-81.
- Cohen SD, Norris L, Acquaviva K, Peterson RA, Kimmel PL. Screening, diagnosis, and treatment of depression in patients with end-stage renal disease. *Clin J Am Soc Nephrol* 2007;2:1332-42.
- Craig JC, Barratt A, Cumming R, Irwig L, Salkeld G. Feasibility study of the early detection and treatment of renal disease by mass screening. *Intern Med J* 2002;32(1-2):6-14.
- Doogue MP, Polasek TM. Drug dosing in renal disease. *Clin Biochem Rev* 2011;32(2):69-73.
- Foley RN, Murray AM, Li S et al. Chronic kidney disease and the risk for cardiovascular disease, renal replacement, and death in the United States medicare population, 1998 to 1999. *J Am Soc Nephrol* 2005;16:489-95.
- Girling JC. Re-evaluation of plasma creatinine concentration in normal pregnancy. *J Obstet Gynaecol* 2000;20(2):128-31.
- Gul A, Aoun N, Trayner EM. Why do patients sleep on dialysis? *Seminars in Dialysis* 2006;19:152-7.
- Hallan SI, Ritz E, Lydersen S, Romundstad S, Kvenild K, Orth SR. Combining GFR and albuminuria to classify CKD improves prediction of ESRD. *J Am Soc Nephrol* 2009;20:1069-77.
- Hanly P. Sleep apnoea and daytime sleepiness in end-stage renal disease. *Seminars in Dialysis* 2004;17:109-14.
- Hedayati SS, Minhajuddin AT, Toto RD, Morris DW, Rush AJ. Prevalence of major depressive episode in CKD. *Am J Kidney Dis* 2009;54:424-32.
- Hedayati SS, Finkelstein FO. Epidemiology, diagnosis, and management of depression in patients with CKD. *Am J Kidney Dis* 2009;54(4):741-52.
- Imai E, Horio M, Nitta K et al. Estimation of glomerular filtration rate by the MDRD study equation modified for Japanese patients with chronic kidney disease. *Clin Exp Nephrol* 2007 March 1;11(1):41-50.
- Johnson DW. Evidence-based guide to slowing the progression of early renal insufficiency. *Intern Med J* 2004;34(1-2):50-7.
- Keith DS, Nichols GA, Gullion CM, Brown JB, Smith DH. Longitudinal follow-up and outcomes among a population with chronic kidney disease in a large managed care organization. *Arch Intern Med* 2004;22;164(6):659-63.
- Levey AS, Eckardt K-U, Tsukamoto Y et al. Definition and classification of chronic kidney disease: A position statement from Kidney Disease: Improving Global Outcomes (KDIGO). *Kidney International* 2005;67:2089-100.
- Levey AS, Stevens LA, Schmid CH et al. A new equation to estimate glomerular filtration rate. *Ann Intern Med* 2009;150:604-12.
- Levin A, Hemmelgarn B, Culleton B et al. Guidelines for the management of chronic kidney disease. *CMAJ* 2008;179(11):1154-62.

Ma Y-C, Zuo L, Chen J-H et al. Modified glomerular filtration rate estimating equation for Chinese patients with chronic kidney disease. *J Am Soc Neph* 2006;17:2937-44.

Mathew TH. How to treat: Microscopic Haematuria. *Australian Doctor* 2007;27-34.

Mathew TH, Johnson DW, Jones GRD, for the Australasian Creatinine Consensus Working Group. Chronic kidney disease and automatic reporting of estimated glomerular filtration rate: revised recommendations. *Med J Aust* 2007;187(8):459-63.

Methven S, MacGregor MS, Traynor JP, Hair M, O'Reilly DSJ, Deighan CJ. Comparison of urinary albumin and urinary total protein as predictors of patient outcomes in CKD. *Am J Kidney Dis* 2010;57(1):21-8.

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

Weiner ME, Tighiouar H, Amin M et al. Chronic kidney disease as a risk factor for cardiovascular disease and all-cause mortality: A pooled analysis of community-based studies. *J Am Soc Nephrol* 2004 May;15(5):1307-15.

White SL, Polkinghorne KR, Atkins RC, Chadban SJ. Comparison of the prevalence and mortality risk of CKD in Australia using the CKD Epidemiology Collaboration (CKD-EPI) and Modification of Diet in Renal Disease (MDRD) Study GFR estimating equations: The AusDiab (Australian Diabetes, Obesity and Lifestyle) Study. *Am J Kidney Dis* 2010;55(4):660-70.

White SL, Yu R, Craig JC, Polkinghorne KR, Atkins RC, Chadban SJ. Diagnostic accuracy of urine dipsticks for detection of albuminuria in the general community. *Am J Kidney Dis* 2011;58(1):19-28.

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

## Australian and International Guidelines

---

Kidney Health Australia Caring for Australasians with Renal Impairment (KHA-CARI) Guidelines. <http://www.cari.org.au>. [See Early Chronic Kidney Disease, Cardiovascular Risk Factors, Biochemical and Haematological Targets].

Chadban S, Howell M, Twigg S et al. National evidence based guideline for diagnosis, prevention and management of chronic kidney disease in type 2 diabetes. Diabetes Australia and the NHMRC, Canberra; 2009.

Crowe E, Halpin D, Stevens P, Guideline Development Group. Early identification and management of chronic kidney disease: summary of NICE guidance. *BMJ* 2008;337 (a1530):812-4.

Diabetes Australia. Diabetes Management in General Practice 17th edition 2011/12. Diabetes Australia; 2011.

Halpin D, Stevens P, Bakhshi L et al. Chronic kidney disease: National clinical guideline for early identification and management in adults in primary and secondary care. London, UK: Royal College of Physicians; 2008.

Moe S, Drueke T, Cunningham J, et al. Definition, evaluation, and classification of renal osteodystrophy: A position statement from Kidney Disease: Improving Global Outcomes (KDIGO). *Kidney International*. 2006;69:1945-1953.

National Aboriginal Community Controlled Health Organisation. National Guide to a preventive health assessment in Aboriginal and Torres Strait Islander Peoples. Royal Australian College of General Practitioners, Melbourne, Australia; 2005.

National Heart Foundation of Australia and the Cardiac Society of Australia and New Zealand. Position statement on lipid management. *Heart Lung and Circulation* 2005;14:275-91.

National Kidney Foundation. K/DOQI clinical practice guidelines for chronic kidney disease: evaluation, classification and stratification. *Am J Kidney Dis* 2002;39(2 Suppl 1):S1-S266.

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

National Vascular Disease Prevention Alliance. Guidelines for the assessment of absolute cardiovascular disease risk, 2009.

Royal Australian College of General Practitioners. Smoking, Nutrition, Alcohol and Physical Activity (SNAP): A population health guide to behavioural risk factors in general practice. South Melbourne, Victoria: The Royal Australian College of General Practitioners; 2004.

Royal Australian College of General Practitioners. Guidelines for preventive activities in general practice (7th edition). The Royal Australian College of General Practitioners, South Melbourne, Australia; 2009.

Scottish Intercollegiate Guidelines Network (SIGN). Diagnosis and management of chronic kidney disease: A national clinical guideline. Edinburgh, UK: Scottish Intercollegiate Guidelines Network; 2008.



## CKD Resources

### Kidney Health Australia

[www.kidney.org.au](http://www.kidney.org.au)

(03) 9674 4300

1800 543 639 – Kidney  
Health Information  
Service Line

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

Programs available to assist health professionals include:

- Face-to-face and online health professional education programs (accredited with RACGP, ACCRM, RCNA)
- Fact sheets
- eGFR resources
- Access to reports and publications

### Kidney Health Australia – Caring for Australasians with Renal Impairment (KHA-CARI)

[www.cari.org.au](http://www.cari.org.au)

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

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

Guidelines available to download online.

### Renal Drug Reference Guide

[www.renaldrugreference.com.au](http://www.renaldrugreference.com.au)

Australian resource focusing on drug therapy in people with CKD. Virtual tours and copies available to order online.

### Renal Resource Centre

[www.renalresource.com](http://www.renalresource.com)

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

## Internet Resources

### Vascular Disease Non-Government Organisations

[www.diabetesaustralia.com.au](http://www.diabetesaustralia.com.au)

[www.heartfoundation.org.au](http://www.heartfoundation.org.au)

[www.strokefoundation.com.au](http://www.strokefoundation.com.au)

### Aboriginal and Torres Strait Islander peoples

[www.naccho.org.au](http://www.naccho.org.au)

[www.kidney.org.au/KidneyDisease/IndigenousResources/  
tabid/770/Default.aspx](http://www.kidney.org.au/KidneyDisease/IndigenousResources/tabid/770/Default.aspx)

[www.healthinfont.ecu.edu.au](http://www.healthinfont.ecu.edu.au)

### Travel on Dialysis

[www.kidney.org.au/ForPatients/DUGDialysisUnitGuide/  
tabid/607/Default.aspx](http://www.kidney.org.au/ForPatients/DUGDialysisUnitGuide/tabid/607/Default.aspx)

### General Practice Professional Organisations

[www.racgp.org.au/](http://www.racgp.org.au/)

[www.acrrm.org.au/](http://www.acrrm.org.au/)

### Nephrology Professional Organisations

[www.nephrology.edu.au/](http://www.nephrology.edu.au/)

[www.asn-online.org/](http://www.asn-online.org/)

## Abbreviations

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ACE inhibitor	Angiotensin-converting enzyme inhibitor
ACRRM	Australian College of Rural and Remote Medicine
ACR	Albumin: creatinine ratio
ALP	Alkaline phosphatase
ARB	Angiotensin II receptor blocker
BMI	Body mass index
BP	Blood pressure
BSL	Blood sugar level
CARI	Caring for Australasians with Renal Impairment
CKD	Chronic kidney disease
CKD-EPI	Chronic Kidney Disease Epidemiology Collaboration
CPAP	Continuous positive airway pressure
CrCl	Creatinine clearance
CVD	Cardiovascular disease
ECG	Electrocardiogram
eGFR	Estimated glomerular filtration rate
ESA	Erythropoiesis stimulating agents
ESKD	End stage kidney disease
GFR	Glomerular filtration rate
KCAT	Kidney Check Australia Taskforce
KDIGO	Kidney Disease Improving Global Outcomes
K/DOQI	Kidney Disease Outcomes Quality Initiative
KHA	Kidney Health Australia
NHMRC	National Health and Medical Research Council
NSAIDs	Non-steroidal anti-inflammatory drugs
PBS	Pharmaceutical benefits scheme
PCR	Protein:creatinine ratio
PTH	Parathyroid hormone
RACGP	Royal Australian College of General Practitioners
RBC	Red blood cells
RCNA	Royal College of Nursing, Australia
RLS	Restless legs syndrome
SBP	Systolic blood pressure
Spiral CT	Spiral computed tomography
TSAT	Transferrin saturation
WC	Waist circumference





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### 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 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.

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## Treatment targets for people with CKD

Parameter	Target	Treatment and effects on systolic BP
<b>Lifestyle Factors</b>		
Smoking	Cease smoking	Lifestyle modification
Nutrition	Dietary salt intake $\leq 100$ mmol/day (or 6 g salt per day)	Lifestyle modification ~ SBP reduction = 2-8 mmHg
Alcohol	Moderate alcohol consumption only ( $\leq 2$ standard drinks on any day)	Lifestyle modification ~ SBP reduction = 2-4 mmHg
Physical activity	>30 mins physical activity/day ~ SBP reduction = 4-9 mmHg	Lifestyle modification
Weight	BMI 18.5 – 24.9 kg/m <sup>2</sup> WC males $\leq 94$ cm ( $\leq 90$ cm in Asian populations) WC females $\leq 80$ cm	Lifestyle modification ~ SBP reduction = 5-20 mmHg/10 kg loss
<b>Lifestyle Factors</b>		
Blood pressure	$\leq 140/90$ mmHg, or $\leq 130/80$ mmHg if albuminuria present (urine ACR $>3.5$ mg/mmol in females and $>2.5$ mg/mmol in males)	Lifestyle modification ACE inhibitor or ARB
Albuminuria	$>50\%$ reduction of baseline value	ACE inhibitor or ARB
Cholesterol	Total $<4.0$ mmol/L LDL $<2.5$ mmol/L	Dietary advice Statins
Blood glucose (for people with diabetes)	HbA1c $<7.0\%$	Lifestyle modification Oral hypoglycaemics Insulin

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



### Clinical Tip

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



## Stages of CKD

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

## Goals of management

<p>Investigations to exclude treatable kidney disease Reduce progression of kidney disease</p> <p>Assessment of absolute cardiovascular risk</p> <p>Avoidance of nephrotoxic medications or volume depletion</p>	<p>Investigations to exclude treatable kidney disease Reduce progression of kidney disease</p> <p>Reduce cardiovascular risk</p> <p>Avoidance of nephrotoxic medications or volume depletion</p>	<p>Investigations to exclude treatable kidney disease Reduce progression of kidney disease</p> <p>Reduce cardiovascular risk</p> <p>Avoidance of nephrotoxic medications or volume depletion</p>
	<p>Early detection and management of complications</p> <p>Adjustment of medication doses to levels appropriate for kidney function</p> <p>Appropriate referral to a Nephrologist when indicated</p>	<p>Early detection and management of complications</p> <p>Adjustment of medication doses to levels appropriate for kidney function</p> <p>Appropriate referral to a Nephrologist when indicated</p>
	<p>Prepare for dialysis or pre-emptive transplant if eGFR &lt;30 mL/min/1.73m<sup>2</sup></p> <p>Discuss advanced care directive if dialysis inappropriate</p> <p>Multidisciplinary team involvement</p>	