

# Mate tākihi ukiuki

## Making a difference in chronic kidney disease

### Part 1: Catching renal impairment early

He matenga ohore, he wairua uiui, wairua mutunga kore

*The grief of a sudden, untimely death will never be forgotten*

Key reviewers:

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#### Key concepts

- Chronic kidney disease (CKD) and end-stage renal failure is much more common in Māori, Pacific, Asian and people from the Indian subcontinent
- CKD is a silent condition but it can be readily detected with eGFR and urinalysis
- Early intervention gives the opportunity to slow progression to end-stage renal disease and reduce cardiovascular risk

[www.bpac.org.nz](http://www.bpac.org.nz) keyword: earlyrenal

Chronic kidney disease (CKD) is the modern term for chronic renal impairment, diagnosed using estimated glomerular filtration rates (eGFR) and markers of kidney damage, detected with urinalysis, imaging or biopsy (Table 1).

Renal impairment has traditionally been diagnosed using serum creatinine. However serum creatinine does not show an abnormal rise until at least 50% of renal function is lost. Diagnosis is often delayed until symptoms or complications of renal failure develop, missing the opportunity for early protective intervention. Using eGFR facilitates early diagnosis of CKD before any change to serum creatinine.

### Chronic kidney disease is common

CKD is estimated to affect approximately 10% of the population. It appears that Māori, Pacific peoples, Asian people and people from the Indian subcontinent have a higher burden of this disease. Māori with diabetes are three and a half times more likely to have renal failure than non-Māori with diabetes.<sup>1</sup>

Only a small proportion of patients with CKD progress to end-stage renal disease. However Māori and Pacific peoples are much more likely to have end-stage renal disease than other ethnic groups in New Zealand. The higher incidence of diabetic and hypertensive end-stage renal disease in Māori and Pacific peoples cannot be fully explained by the underlying disease prevalence. Other factors that may contribute to this difference in incidence are socio-economic disadvantage, which is a known risk factor for end-stage renal disease, and underutilisation of medical services.

People with end-stage renal disease commonly have a progressive fall in eGFR, marked proteinuria and hypertension. Early recognition and management, and appropriate referral can prevent end-stage renal disease in some cases and greatly improve the outcome for others.<sup>2</sup>

#### Markers of kidney damage include:

- Persistent proteinuria/albuminuria
- Persistent haematuria/ WBC in urine
- Red blood cell casts/dysmorphic cells on urine microscopy
- Ultrasound/other radiological abnormalities

**Table 1:** CKD staging based on eGFR and markers of kidney damage

eGFR (mL/min/1.73 m <sup>2</sup> )	Markers of kidney damage (see box)	CKD staging	Description
>60	No	No chronic kidney disease	Renal function declines with age
	Yes	1 = eGFR >90	Normal kidney function but urine findings or structural abnormalities or genetic trait point to kidney disease
		2 = eGFR 60–89	Mildly reduced kidney function, and other findings (as for stage 1) point to kidney disease
<60	Not required for staging	3 = eGFR 30–59	Moderately reduced kidney function
		4 = eGFR 15–29	Severely reduced kidney function
		5 = eGFR <15, or needing dialysis	Very severe, or end-stage renal disease

It is important to note that CKD is an independent cardiovascular risk factor and more people with CKD die from cardiovascular disease than end-stage renal disease.<sup>3</sup>

Being able to detect CKD early allows:<sup>4</sup>

- Early protective intervention to reduce progression towards end-stage renal disease (CKD stage 5)
- Monitoring and treatment of cardiovascular risk factors to reduce cardiovascular disease
- Monitoring and treatment of complications

### Quick clinical assessment for kidney health check

#### History including:

- Any risk factors, particularly hypertension
- Prescribed, OTC and alternative medication
- Symptoms of cardiovascular disease e.g. breathlessness, oedema, chest pain, claudication
- Symptoms suggestive of underlying systemic diseases such as vasculitis, lupus or myeloma e.g. fever, weight loss, fatigue, general aches and pains

#### Examination:

- Dipstick urine
- Blood pressure
- Weight
- Fluid status (JVP, signs of pulmonary oedema, peripheral oedema)
- Enlarged bladder or kidneys
- Renal bruits
- PR in male with lower urinary tract symptoms

### Targeted testing for chronic kidney disease in New Zealand

Targeted testing should be considered for people with the following risk factors:

- Aged over 50 years
- Hypertension
- Any cardiovascular disease (IHD, chronic heart failure, peripheral vascular disease and cerebral vascular disease)
- Diabetes
- Smoking
- Known personal or family history of kidney disease, including recurrent UTIs and lower urinary tract symptoms
- Māori, Pacific peoples, Asian people and people from the Indian subcontinent
- Long-term treatment with nephrotoxic medication such as lithium, cyclosporin, mesalazine (NSAIDs are not nephrotoxic but use increases the risk of kidney damage)

It is recommended to perform a kidney health check at a minimum of every five years. This should be done annually if diabetes, established cardiovascular disease or CKD is present.

 **Best practice tip:** link a kidney health check with a cardiovascular risk assessment by adding in urinalysis and serum creatinine.

#### Kidney Health Check

The kidney health check includes

- eGFR
- Urinalysis for proteinuria or microalbuminuria
- Blood pressure measurement

Further investigations depend on the results of these tests. Any abnormalities should prompt history-taking and examination.

## eGFR results and action required

Laboratories provide the eGFR automatically when reporting serum creatinine results. If the eGFR is greater than 90 no further action is required unless there is suspicion of kidney disease. Levels less than 90 require urinalysis (Table 2).

The confirmation of a new CKD stage is based on a minimum of two eGFR values taken over three months. For newly diagnosed patients it is important to establish the rate of decline.

**Table 2:** Results of eGFR and required action

eGFR value	Action
>90	“No further action” unless known, or suspected, structural or urinalysis abnormalities.
60–89	Urinalysis required to check for evidence of kidney disease. If negative and no other markers of renal disease “no further action” required. If positive determine CKD stage.
<60	Exclude acute renal failure. Determine CKD stage. Urinalysis required to check for haematuria/proteinuria.

**N.B.** Most laboratories report the eGFR either as >90 mL/min/1.73m<sup>2</sup> or, if less than this, as an exact figure.<sup>5</sup> Some laboratories only give exact figures if the result is less than 60.

If the eGFR is unexpectedly low, best practice is to exclude acute renal failure by a repeat serum creatinine/eGFR. If the patient is obviously ill with rising blood pressure, oedema, proteinuria, haematuria and an unexpected falling eGFR or rising creatinine, discuss urgently with the nephrology team as this may indicate acute kidney damage e.g. glomerulonephritis.

## Urinalysis

Urinalysis can provide indirect evidence of kidney damage. Inflammation or abnormal function of the glomeruli can lead to leakage of red blood cells or protein into the urine, resulting in haematuria or proteinuria.

Urine dipstick testing is recommended as a screening test in general practice. Standard dipstick analysis is adequate to screen for proteinuria and haematuria, but will not detect microalbuminuria unless an albumin specific dipstick is used.

 **Best practice tip:** Most eGFRs in people with risk factors for CKD will be 60–89 and urinalysis will be required to decide if CKD is present. As proteinuria levels may fluctuate during the day an early morning urine for analysis is preferred.

Abnormal results should prompt:

- A mid-stream urine (MSU) looking for red blood cell casts or dysmorphic cells on microscopy or, if nitrates or leucocytes positive, culture for infection

and/or

- Early morning urinalysis for albumin:creatinine ratio (ACR) or protein:creatinine ratio (PCR) (Table 3). ACR and PCR have superseded 24 hour urine collection for quantification of proteinuria.

**Table 3:** Further tests required after dipstick urine

Dipstick result	MSU	ACR	PCR
No abnormality		✓ if diabetic or hypertensive	
Haemoglobin/blood +ve	✓		
Albumin +ve (albumin specific dipstick)	✓	✓ if diabetic or hypertensive	
Protein +ve	✓		✓

## eGFR is an estimate

Although eGFR is now the basis of diagnosis of CKD it is only an estimate. The eGFR is calculated from the serum creatinine, using the four-variable Modification of Diet in Renal Disease (MDRD) equation, based on age and a standard adult body surface area. Results are reported as mL/min/1.73m<sup>2</sup>. As it does not allow for any variation in body weight, a significant error is possible.

eGFR is not valid in pregnant women or in children (<18 years), nor accurate if the serum creatinine is changing rapidly as occurs in acute renal failure.

Creatinine level is dependant on muscle mass and the eGFR is most likely to be inaccurate in people at extremes of body type, e.g. reassuringly high in people with reduced muscle mass such as the frail elderly, amputees, people with chronic liver disease or on low protein diets (vegan).

The MDRD equation was originally validated in the United States. Although it has not been validated for Māori and Pacific peoples, Asian people or those from the Indian subcontinent, the eGFR is still recommended for screening for CKD in these populations.

An alternative calculation, that uses the individual's weight as an approximation of lean body mass, is the Cockcroft-Gault equation for calculating creatinine clearance as mL/min (the proxy measure for glomerular filtration rate). This equation is preferred for drug dosage adjustment, although it has similar disadvantages as the MDRD equation for people at extremes of body type.

Creatinine clearance mL/min =

$$\frac{(140 - \text{age}) \times \text{weight}(\text{kg}) \times 0.85(\text{in females})}{815 \times \text{serum creatinine}(\text{mmol/L})}$$

815 x serum creatinine(mmol/L)

Transient proteinuria or haematuria is not unusual. Urine dipstick blood or protein that does not resolve should be followed up. Persistent haematuria requires referral to either urology or nephrology.

## Other investigations

Ultrasound is the optimal first line test for imaging the renal tract in patients with CKD and identifies obstructive uropathy, renal size and symmetry, renal scarring and polycystic disease. It is recommended where there is lower urinary tract symptoms, eGFR < 60 mL/min/1.73m<sup>2</sup>, progressively falling eGFR or refractory hypertension (blood pressure >150/90 mmHg despite three or more antihypertensives).

## Further reading

The following guidelines were considered in the development of this article:

- Kidney Health New Zealand (KHNZ). Chronic kidney disease (CKD) management in general practice: summary guide. KHNZ 2009.

Available from [www.kidneys.co.nz](http://www.kidneys.co.nz)

- Kidney Health Australia (KHA). Chronic kidney disease (CKD) management in general practice: guidance and clinical tips to help identify, manage and refer CKD in our practice. KHA, Melbourne 2007.

Available from [www.kidney.org.au](http://www.kidney.org.au) or [www.racgp.org.au](http://www.racgp.org.au)

- Scottish Intercollegiate Guidelines Group (SIGN). Diagnosis and management of chronic kidney disease: a national clinical guideline. SIGN, Edinburgh 2008.

Available from [www.sign.ac.uk](http://www.sign.ac.uk)

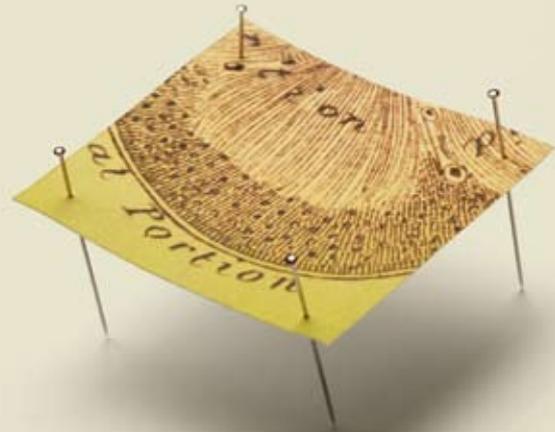
## Definitions of microalbuminuria and proteinuria

Detection of an increase in protein excretion has both diagnostic and prognostic value in the initial detection and confirmation of renal disease.

Persistent higher-risk microalbuminuria is defined by an early morning ACR of  $>2.5$  mg/mmol in men or  $>3.5$  mg/mmol in women on two or more occasions.

In non-diabetic CKD significant proteinuria is regarded as  $> 0.3$  g/day on two separate occasions. This is equivalent to PCR  $>30$  mg/mmol.

 **Best practice tip:** a PCR of 100 mg/mmol is equivalent to daily protein excretion of 1 g/24 hours.



- National Institute for Clinical Excellence (NICE). Chronic kidney disease: national clinical guideline for early identification and management in adults in primary and secondary care. NICE, London, 2008. Clinical Guideline 73.

Available from [www.nice.org.uk](http://www.nice.org.uk)

- Rosenberg M, Kalda R, Kasiulevicius V, Lember M. Management of chronic kidney disease in primary health care: position paper of the European Forum for Primary Care. Qual Primary Care 2008;16:279-94.

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## References

1. Ministry of Health. Māori Health. Health status indicators: Diabetes. 2002/03 New Zealand Health Survey. Available from: [www.Māorihealth.govt.nz/moh.nsf/indexma/diabetes](http://www.Māorihealth.govt.nz/moh.nsf/indexma/diabetes) (Accessed July 2009).
2. Richards N, Harris K, Whitfiels M, et al. Primary care-based disease management of chronic kidney disease (CKD), based on estimated glomerular filtration rate (eGR) reporting, improves patient outcomes. Nephrol Dial Transplant 2008;23:549-55.
3. Go AS, Chertow GM, Fan D, et al. Chronic kidney disease and the risks of death, cardiovascular events, and hospitalisation. NEJM 2004;351(13):1296-1305.
4. Gomez GB, de Lusigan S, Gallagher H. Chronic kidney disease: a new priority for primary care. Brit J Gen Pract 2006;908-10.
5. Saleem M, Florkowski C for the Australasian Creatinine Consensus Working Group. Reporting of estimated glomerular filtration rate (eGFR) in New Zealand – what are the clinical laboratories doing? N Z Med J 2006;119(1246).

# Mate tākihi ukiuki

## Making a difference in chronic kidney disease

### Part2: Management

Key reviewers:

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**Māna anō e whakamāui ake** – *May the person recover. The expression whakamāui comes from a legend in which Māui was waylaid and almost killed. However he restored himself to health using incantations.*

#### Key concepts:

- ACE inhibitors and angiotensin 2 receptor blockers (ARBs) are indicated in all patients with a stable eGFR less than 60 mL/min/1.73 m<sup>2</sup> with hypertension, or with proteinuria or diabetes irrespective of the blood pressure
- Medicines management is vital – check the recommended doses in renal impairment and avoid NSAIDs if possible

## Initial Management

Management is dependent on the CKD stage, the level of cardiovascular risk and the presence of any red flags or indications for referral (Table 1).

**Table 1:** Red flags and referrals (adapted from McKie et al, 2006)<sup>1</sup>

<b>RED FLAGS</b> urgent/same day referral	<ul style="list-style-type: none"> <li>▪ eGFR &lt;15 (CKD level 5) newly detected – to Nephrology</li> <li>▪ Possible acute glomerulonephritis: unwell, dehydrated, rapidly rising creatinine, increasing oedema and blood pressure, proteinuria and haematuria – to Nephrology</li> <li>▪ Hyperkalaemia &gt;7 mmol/L – to Physician</li> <li>▪ Acute “obstructive” renal failure – to Urologist</li> </ul>
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Referral	Indications
<b>Urology</b>	<ul style="list-style-type: none"> <li>▪ Abdominal/loin mass on examination or ultrasound</li> <li>▪ Evidence of prostate cancer</li> <li>▪ Renal colic</li> <li>▪ Painless macroscopic haematuria (non-UTI)</li> </ul>
<b>Nephrology</b>	<ul style="list-style-type: none"> <li>▪ eGFR 15–29 (CKD level 4)</li> <li>▪ Progressively falling eGFR e.g. &gt;4 mL/min/1.73m<sup>2</sup> over a year</li> <li>▪ Suspected multisystem disease such as rheumatoid arthritis, SLE, other vasculitides</li> <li>▪ Possible nephrotic syndrome: gross proteinuria, oedema, hypoalbuminaemia</li> <li>▪ Persistent abnormalities of urinalysis: PCR &gt;100 mg/mmol (some clinicians recommend referral at &gt;50 mg/mmol); sterile pyuria; microscopic/macroscopic haematuria, especially if with proteinuria</li> <li>▪ Blood pressure uncontrolled to 150/90 mmHg despite three or more antihypertensives</li> <li>▪ CKD complications e.g. Hb &lt;100 g/L with normal iron stores or abnormal potassium/calcium/phosphate/Alk Phos/PTH)</li> </ul>

## Managing Change

Māori knowledge of chronic kidney disease comes largely from the experiences of whānau members – those who have died and those who have end-stage renal disease. Memories are of tangihanga (funerals) and of loss.

Some Māori who have been diagnosed with chronic kidney disease may avoid contact with any health service. Once they are “lost to the system” it is very difficult for a GP to assist in the management of their care. However it is important not to stereotype Māori as many are fully compliant with treatment. GPs are well equipped to provide important health information to whānau and to assist in managing this disease. However, in order for information

to be heard and understood, a connection must be made. The development of a trusting therapeutic relationship with the patient and key whānau members is important.

Listen to the patient and whānau to understand how they feel. Acknowledging whānau experiences and loss can help develop a successful relationship so that together the GP and whānau can plan the next steps – bearing in mind that how the journey is to be walked and shaped will be determined by the patient and whānau.

Accepting and living with this type of significant change is probably one of the most difficult requests we make of our patients.

## Indications for referral

Most patients with stable or slowly declining eGFR, minor proteinuria (PCR <30 mmol/mL) with no haematuria, and controlled blood pressure can be managed in general practice, until the CKD declines to the level where decisions need to be made about dialysis (CKD stage 4 onwards).

All patients with an eGFR of 30 or less should be considered for referral to a Nephrologist for guidance with management whether considering renal replacement therapy (dialysis) or palliative care.

Key facts to include in the referral are: any known risks for CKD; lower/upper urinary tract symptoms; medications; oedema, masses and other relevant findings; blood pressure; quantification of proteinuria; current bloods; renal ultrasound results (recent); and all previous serum creatinine results (eGFR) listed by date.

## Tests required for monitoring

The tests required for monitoring are dependent on CKD stage (Table 2) and clinical appropriateness.

**The six key parameters** to monitor for every patient with CKD are:

1. Annual cardiovascular risk
2. Blood pressure
3. Weight
4. Urinalysis (PCR should be measured annually if there is persistent proteinuria)
5. Serum creatinine/eGFR
6. Potassium

It is recommended that cardiovascular risk assessment should be done annually with all CKD stages.

**Table 2:** Recommended ongoing investigations depending on CKD stage

CKD stage	Minimum frequency	Investigations
<b>1 &amp; 2</b>	Annually	Key parameters
<b>3</b>	Three monthly then six monthly if stable	Key parameters + CBC Calcium Phosphate Alk Phos PTH (at diagnosis and if calcium, phosphate or Alk Phos levels become abnormal)
<b>4 &amp; 5</b>	Three monthly then six monthly if stable	Key parameters + CBC Calcium Phosphate Alk Phos PTH (six monthly)

 **Best practice tip:** As eGFR falls it is necessary to adjust the dose of drugs that are renally excreted and to avoid nephrotoxic medications. As the eGFR drops below 60 mL/min/1.73 m<sup>2</sup> link the regular laboratory tests with a medicines review. Recalculate Cockcroft-Gault creatinine clearance with the new creatinine result and review all medication.

## Ongoing management

The main aims of ongoing care of CKD are:

1. Early protective intervention to reduce progression towards end-stage renal disease
2. Monitoring and treatment of cardiovascular risk factors
3. Monitoring and treatment of complications

### Maintaining blood pressure below target levels is a key goal

CKD can cause and aggravate hypertension which can accelerate the deterioration of renal function. Reducing blood pressure to target levels is one of the most important goals in the management of CKD. The target level is less than 130/80 mmHg, or less than 125/75 mmHg in people with diabetes or if the PCR is greater than 100 mg/mmol.

With the exception of salt wasting nephropathies e.g. tubular damage after obstruction, salt retention is a major factor in the hypertension related to CKD. Dietary reduction of sodium intake to <80 mmol/day (~ 1 tsp salt) is recommended. Salt reduction can achieve BP reductions equivalent to a single antihypertensive agent. The majority of dietary sodium intake is derived from processed food including bread and dietetic advice is usually required.

ACE inhibitors and diuretics are the main stay of antihypertensive therapy in CKD. Hypertension may be difficult to control and three to four medications are frequently required in CKD stages 3–5.

### Start ACE inhibitor or ARB unless contraindicated

Proteinuria is a sign of renal damage. Increasing amounts of protein in the urine correlates directly with an increased rate of progression to end-stage kidney disease. ACE inhibitors and ARBs significantly reduce proteinuria and improve renal and cardiovascular outcomes.

ACE inhibitors are therefore recommended as first-line agents for the control of blood pressure and for all patients

## Ongoing haematuria

The presence of three or more erythrocytes per high-power field in at least two of three urine samples is considered confirmatory of ongoing haematuria. This requires further investigation and referral.

Macroscopic or microscopic haematuria may be a sign of urinary tract malignancy. In patients with risk factors, such as smoking and older age at presentation, urological investigation is the most appropriate first step.

In the younger age group nephrological and urological aetiologies need to be considered. If in doubt urine cytology is a useful screening tool. When associated with proteinuria and an abnormal eGFR, a medical/nephrological evaluation is indicated.<sup>2</sup>



with proteinuria (PCR >100 mg/mmol) at CKD stage 3 or worse.

People with diabetes and CKD should be prescribed an ACE inhibitor or ARB irrespective of their blood pressure reading.

### Tips for reducing salt in the diet

- Look for “no salt added” labels in food
- Use fresh foods rather than canned varieties
- Rinse canned foods e.g. tuna to remove salt
- Do not add salt to cooking
- Avoid “instant” or convenience foods which often have a high salt content

### Initiating ACE inhibitors and ARBs

ACE inhibitors and ARBs may cause a significant drop in eGFR and rise in serum potassium and creatinine. Serum potassium and creatinine should be monitored after initiation or when doses are changed (after one to two weeks). Any concerns can be discussed with a Nephrologist.

Most patients with CKD (especially on an ACE inhibitor or ARB) will have mild hyperkalaemia (5.2 – 5.8 mmol/L). This is not an indication to stop their ACE inhibitor. Review dietary potassium intake and discontinue other drugs known to promote hyperkalaemia. Medications that may be associated with hyperkalaemia include potassium supplements, potassium sparing diuretics, NSAIDs, Cox-2 inhibitors, digoxin, lithium and trimethoprim.<sup>3</sup>

The fall in eGFR should stabilise to be less than 15% from baseline (or 20% increase in serum creatinine). If this is exceeded consider volume depletion or concurrent medication, especially NSAIDs, Cox-2 inhibitors and diuretics. If no other cause is found reduce the dose to tolerable levels e.g. halve.

Renal artery stenosis requiring intervention is rare. Where serum creatinine rises rapidly (a doubling or more over three to five days after commencement of an ACE inhibitor) consider urgent discussion/referral to nephrology. Most cases will be managed with medical therapy including an ACE inhibitor.

### Cardiovascular risk reduction and diabetic control

The presence of CKD is one of the strongest risk factors for cardiovascular disease. Be aware that the Framingham tables significantly underestimate risk when CKD is present. All people with CKD should undergo cardiovascular disease risk factor modification, especially for smoking and hypercholesterolaemia.

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

### Medicine management: stop unsafe medication

The clearance of many drugs and their metabolites depends on adequate renal function. As eGFR falls below 60 mL/min it is necessary to adjust the dose of drugs that are renally excreted and avoid nephrotoxic medications. If possible NSAIDs and COX-2 inhibitors should be avoided. A combination that can be fatal in CKD is the “triple whammy” of an NSAID, ACE inhibitor and a diuretic.

 **Best practice tip:** Warn patients with CKD on antihypertensives to:

- Stop diuretics (and NSAIDs, if using them) if they become unwell with diarrhoea and/or vomiting
- Discuss what to use for pain relief with a pharmacist or GP

The Cockcroft-Gault equation should be used for calculating creatinine clearance (mL/min) when adjusting medications in renal impairment (Table 3). Dosages of all medications should be checked with prescribing guidelines e.g. BNF Appendix 3 on renal impairment, datasheets from Medsafe and MIMS.

### Infections are a major cause of death in people with end-stage renal disease

Infections are a major cause of death in people with end-stage renal disease, second only to cardiovascular disease. Patients with CKD are immunocompromised but can be vaccinated successfully with augmented immunisation regimens.

Vaccination against influenza and pneumococcus are recommended from CKD stage 3. Influenza vaccine, in particular, seems to provide adequate protection with standard dosing regimens, while pneumococcus requires an augmented regimen with re-vaccination every three to five years.

Hepatitis B vaccination is recommended for those expected to go on to dialysis (consider from CKD stage 4) and requires booster doses when the antibody titre drops below 10 UI/L.<sup>5,6</sup>

## Complications of CKD

The rate of complications increases as the eGFR drops below 60 mL/min/1.73 m<sup>2</sup>.

Key complications that require referral are:

- Mineral bone disorders (classically with raised serum phosphate, alk phos and PTH and low calcium and vitamin D levels)
- CKD-related anaemia. Exclude other causes of Hb <100 g/L e.g. low ferritin, TSH. Local renal units may have arrangements for shared care with general practice.<sup>7</sup>

Others complications, as renal function continues to decline, include:

- Anorexia, nausea and poor food intake resulting in malnourishment
- Metabolic acidosis (low serum bicarbonate)
- Hyperkalaemia
- Restless legs syndrome

- Sleep apnoea
- Depression

## Management of uraemia in end-stage renal disease (CKD stage 5)

End-stage renal disease (CKD stage 5), is associated with very high levels of serum urea and creatinine from the accumulation of breakdown products of protein metabolism. The symptoms of uraemia include anorexia, nausea, vomiting, pruritis, lethargy, muscle twitching, muscle cramps, polyuria, peripheral oedema and dyspnoea. Pain is a common symptom in this group of patients, often from co-morbidities. Hiccoughs, confusion, convulsions and coma are rarely seen and indicate very severe renal disease.

Ideally patients should be referred to nephrology at CKD stage 4, well before the onset of uraemic symptoms, for discussion of the available choices, including consideration of and planning for possible renal replacement therapy (dialysis or kidney transplant).

**Table 3:** Some commonly prescribed drugs that require dose adjustment in renal impairment (adapted from Faull et al, 2007).<sup>4</sup>

Class	Examples
<b>Antibiotics/antifungals</b>	Aminoglycosides (e.g. gentamicin), flucloxacillin, ciprofloxacin, fluconazole
<b>Antivirals</b>	Aciclovir
<b>Anticoagulants</b>	Low molecular weight heparins (e.g. enoxaparin)
<b>Cardiac drugs</b>	Digoxin, sotalol, atenolol
<b>Diuretics</b>	If creatinine clearance is less than 30 mL/min: – Use potassium-sparing diuretics with caution due to risk of hyperkalaemia – Thiazide diuretics have limited efficacy at standard doses, consider swapping to frusemide
<b>Opioids</b>	Morphine, codeine, pethidine (due to risk of accumulation of active metabolites)
<b>Psychotropics/ anticonvulsants</b>	Gabapentin, haloperidol, lithium, risperidone
<b>Hypoglycaemic drugs</b>	Metformin, glibenclamide, insulin
<b>Drugs for gout</b>	Allopurinol, colchicine
<b>Others</b>	NSAIDs, methotrexate, penicillamine

Some people reaching CKD stage 5 will be clinically unsuitable for dialysis. This is generally due to functional decline, frailty and/or coexisting conditions, all of which make successful dialysis therapy very unlikely. Some individuals choose not to have dialysis for personal reasons or to withdraw from the dialysis programme.

A patient with end-stage renal disease who chooses not to have dialysis has an average survival of six to eight months.<sup>8</sup> Symptom management is challenging in this group and referral to the local palliative care team, in conjunction with the nephrology service, may be appropriate.<sup>9</sup> Māori providers may also be able to assist.

In general if a patient does not undergo dialysis:

- A low protein diet will help control gastrointestinal symptoms—advice from a dietitian is recommended

- Fluid control should be strict to avoid pulmonary oedema
- Avoid unnecessary medications
- Seek specialist advice on appropriate drugs and dose modification for symptom management

## Further reading

The following guidelines were considered in the development of this article:

- Kidney Health New Zealand (KHNZ). Chronic kidney disease (CKD) management in general practice: summary guide. KHNZ 2009. Available from [www.kidneys.co.nz](http://www.kidneys.co.nz)
- Kidney Health Australia (KHA). Chronic kidney disease (CKD) management in general practice:

## Patient support and information

As with any chronic disease, people with CKD need to have their ideas, concerns and expectations explored.<sup>10</sup> Many patients will believe “kidney disease” means dialysis and a shortened life. The following is a selection of appropriate educational material:

“Chronic Kidney Disease: What it is - What it means” provides information relevant to the majority of patients. From the RCGP and National Kidney Foundation (UK). Available from:

[www.renal.org/pages/modules/download\\_gallery/dlc.php?file=285](http://www.renal.org/pages/modules/download_gallery/dlc.php?file=285)

“Chronic renal failure and its progression” plus other articles from the Renal Unit of the Royal Infirmary of Edinburgh (EdREN) is more suited to those at higher risk of progressive renal disease. Available from:

<http://renux.dmed.ed.ac.uk/EdREN/EdRenINFObits/CRFLong.html>

“Kidney patient guide” is a web guide to the function of the kidney, kidney failure and its treatment including animations. Available from:

[www.kidneypatientguide.org.uk/site/physical.php](http://www.kidneypatientguide.org.uk/site/physical.php)

Kidney Society (Auckland) provides information on end-stage renal disease and its treatment. Available from:

[www.kidneysociety.co.nz/home/about-kidneys-and-kidney-failure.html](http://www.kidneysociety.co.nz/home/about-kidneys-and-kidney-failure.html)

They also provide a telephone help-line 0800 235 711 (9am to 5pm) and home visits in the Auckland and Northland region.

The New Zealand Kidney Foundation has a series of factsheets mainly on end-stage renal disease, and support centres throughout the country (includes Māori language resources). Ph: 0800 543 639. [www.kidneys.co.nz](http://www.kidneys.co.nz)

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## Rongoā for kidney complaints

There are a number of traditional Rongoā Māori treatments for kidney and urinary complaints. These include Kawakawa (Māori Pepper Tree), Karamu (Coprosma), Manuka (Red tea tree) and Kanuka (White tea tree). The leaves and shoots are generally boiled in water and the liquid ingested.

Ask patients about any Rongoā or other alternative remedies they are taking. It is important to be aware of this so any possible conflict with conventional medicine or treatment can be assessed.

