J R Coll Physicians Edinb 2020; 50: 67–74 | doi: 10.4997/JRCPE.2020.119 REVIEW

A practical guide to diagnosis and assessment of chronic kidney disease for the non-nephrologist

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Abstract

Chronic kidney disease (CKD) is common. People with CKD have a wide range of comorbidities and, therefore, the majority of non-nephrologists will care for people with CKD. This paper aims to provide a brief overview of the diagnosis and management of CKD for the non-nephrologist.

Identifying those with CKD and optimising treatment is essential as CKD has a direct association with adverse patient outcomes. There are modifiable factors where interventions may delay progression of CKD, including: smoking cessation, dietary advice, hypertension management, renin-angiotensin system blockade, glycaemic control and relieving urinary outflow obstruction. Complications, such as renal anaemia, metabolic acidosis, CKD-related mineral bone disease, hyperkalaemia and gout, are best managed in conjunction with nephrology input.

The progression of CKD is often variable and nonlinear, but person-centred intervention can delay progression of CKD, reduce morbidity and mortality, and allow time for preparation for renal replacement therapy, ultimately providing the best possible personalised care.

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Keywords: chronic kidney disease, guidelines, hypertension, proteinuria

Financial and Competing Interests: No conflict of interests declared

Introduction

Chronic kidney disease (CKD) is common with approximately 6% of the UK adult population having an estimated glomerular filtration rate (eGFR) <60 ml/min/1.73 m^2, and the majority of non-nephrologists will care for people with CKD.[1] In contrast, end-stage kidney disease (ESKD) requiring renal replacement therapy is extremely uncommon with an incidence of around 120 per million population in the UK.[2] This paper aims to provide a brief overview of diagnosis and investigation of people with CKD for the non-nephrologist. The first part of the review focuses on the diagnosis of CKD that may be undertaken by the non-nephrologist and the second part provides an overview into how the nephrology team may manage CKD.

Part 1: diagnosis of CKD

Classification of CKD

The international Kidney Disease: Improving Global Outcomes (KDIGO) guidelines recommend classification of CKD based on the cause of CKD, eGFR and albuminuria [measured by albumin:creatinine ratio (ACR)], abbreviated to the 'CGA' classification system.[3] Identifying those with CKD and optimising treatment is important as CKD has a direct association with adverse patient outcomes. Increasing proteinuria and decreasing glomerular filtration rate (GFR) are both associated independently with adverse outcomes, including increased all cause mortality, in particular cardiovascular death, acute kidney injury (AKI) and progression to ESKD.[4] Having both decreased GFR and significant proteinuria multiply that risk, as seen in the heat map in Figure 1.[3]

Who should undergo screening for CKD?

There is no population-based CKD screening programme in the UK. However, it is recommended that people at high risk, including those with conditions associated with CKD, structural abnormalities in their renal tract and those on nephrotoxic medication, should be screened for CKD at least annually, as shown in Table 1.[5]

Diagnosis of CKD

For the majority of people, a diagnosis of CKD is based primarily on the results of blood tests and urine protein testing as outlined in Figure 1, though patients with normal excretory renal function may still have CKD if they have an alternative marker of kidney damage. These include:

- proteinuria
- haematuria of renal origin
- structural renal abnormality
- genetic diagnosis affecting the kidneys, e.g. polycystic kidney disease

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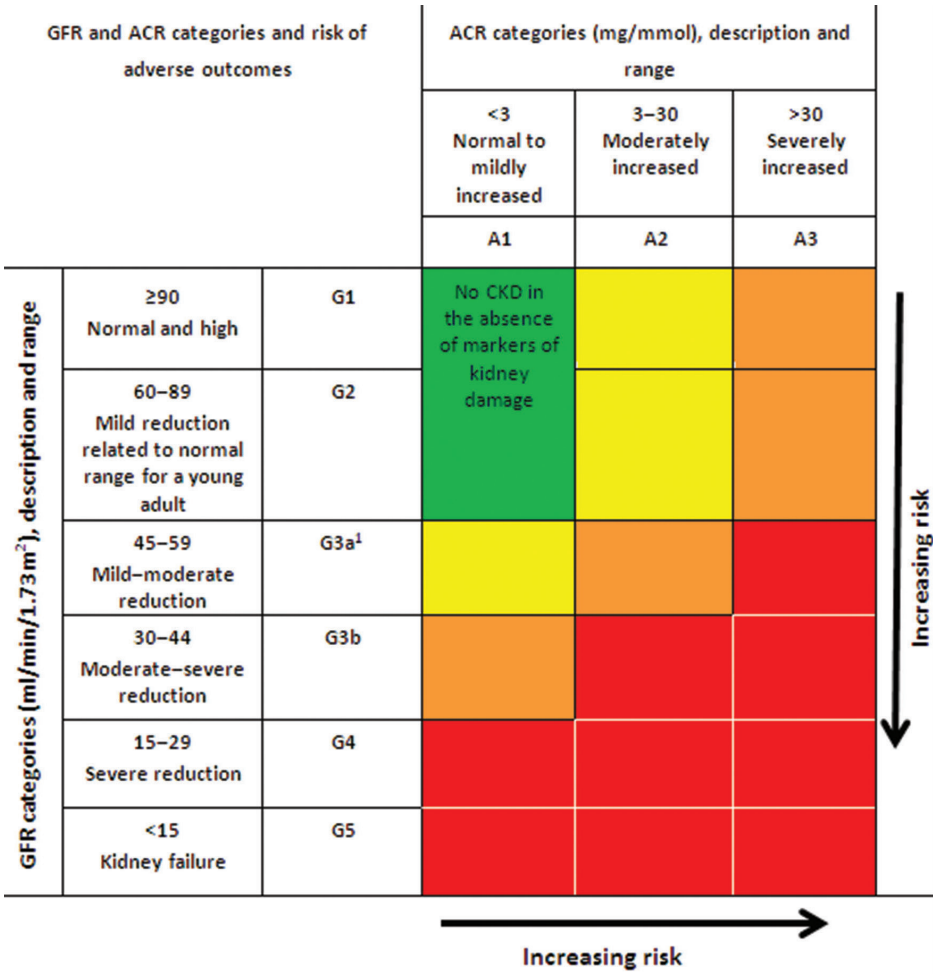


Figure 1 Classification of CKD using GFR and ACR categories. Adapted with permission from Kidney Disease: Improving Global Outcomes (KDIGO).[3]
[1]Consider using eGFR-based cystatin C for patients with CKD G3aA1 (see recommendations 1.1.14 and 1.1.15 from KDIGO Group[3]).
ACR: albumin:creatinine ratio; CKD chronic kidney disease; eGFR: estimated glomerular filtration rate; GFR glomerular filtration rate

- electrolyte abnormalities due to tubular damage
- abnormalities on renal biopsy.

1. Blood tests: U+Es/eGFR

As an initial screening test, serum creatinine and eGFR are appropriate. With the finding of a first abnormal blood test (eGFR <60 ml/min/1.73 m^2), it is important to exclude AKI in the first instance by repeat testing within 14 days.[5] A diagnosis of CKD based on reduced eGFR requires two measurements of eGFR <60 ml/min/1.73 m^2 over 90 days apart. Although helpful for screening those at risk of CKD and initial testing, the use of eGFR does have some caveats and does not always reflect the severity of CKD on an individual basis and can be extremely variable within the same patient. It is less accurate in patients with extremes of muscle mass, such as body builders, and those with above-average muscle mass can be overdiagnosed with CKD. Similarly, those with low muscle mass, for example owing to muscle wasting, malnutrition, amputation or liver disease, may have relatively advanced renal disease with less abnormal biochemistry on initial testing.[6] Cystatin C-based eGFR can be useful in these situations and may be more accurate at assessing their eGFR.[7] This may be recommended by a nephrologist or clinical biochemist but should not be routinely used. The guidance from the National Institute for Health and Care Excellence (NICE) recommend checking cystatin C-based eGFR for those who have an unexpected/isolated finding of eGFR 45–60 ml/min/1.73 m^2 using serum creatinine-based eGFR without other evidence of renal disease to confirm the diagnosis.[5]

2. Urine dip (routine urine examination)

a. Proteinuria

Proteinuria is the strongest predictor of renal risk and is associated with adverse patient outcomes.[8] Quantifying proteinuria is essential as mortality, cardiovascular events and progression of renal disease all increase with increasing levels of proteinuria.

Proteinuria causes both glomerular and tubular damage that through a variety of pathways leads to increased proteinuria and progression of CKD. As mentioned above, it is also independently linked to cardiovascular morbidity and mortality.

Proteinuria should be formally quantified with ACR or protein creatinine ratio (PCR) in people with suspected CKD (or nephrotic syndrome). These ratios are calculated by dividing the urine protein or albumin (measured in milligrams per litre) by the urine creatinine concentration (millimoles per litre) to correct for urine flow rate. There is no benefit of the routine use of 24-hour urine collections that are cumbersome for patients and usually poorly performed.[9] Low-level proteinuria should be repeated to confirm, but levels above ACR 70 mg/mmol do not require this, though levels should be monitored regularly. ACR

Table 1 Who should be tested for evidence of CKD

Patient group	Examples
Those with risk factors for CKD	Diabetes, hypertension, cardiovascular disease, previous acute kidney injury
Structural renal tract disease	Renal stone, benign prostatic hypertrophy, chronic reflux nephropathy
Taking potentially nephrotoxic medication	NSAIDs, ACE inhibitors/ARB, lithium, calcineurin inhibitors, 5-ASA

5-ASA: 5 aminosalicylic acid; ACE: angiotensin converting enzyme; ARB: angiotensin II receptor blockers; CKD: chronic kidney disease; NSAID: nonsteroidal anti-inflammatory drug

is generally used for assessment in diabetic kidney disease, however, ACR and PCR have both been found to be equally effective in predicting outcomes even at low levels.[8]

b. Haematuria

Patients with unexplained *visible haematuria* aged >45 years should initially have urological assessment including imaging to exclude renal tract malignancy unless there is significant AKI or other evidence of intrinsic renal disease.[10]

Assessment of *nonvisible haematuria* (NVH) should be undertaken using reagent strips rather than microscopy. Persistent NVH is defined as two or more positive tests from three consecutive samples of dipstick 1+ or more. Unlike proteinuria, NVH is often not due to an intrinsic renal pathology, but may be caused by urological disease. Referral to urology for exclusion of renal tract malignancy should also be considered for those aged >60 years with persistent NVH plus dysuria or raised white cell count in the absence of urinary tract infection.[10] If urological disease is excluded, people with persistent NVH may have IgA nephropathy and, therefore, should have annual urinalysis (for protein and blood) plus quantification of proteinuria if indicated, and eGFR and blood pressure (BP) checks for as long as the NVH persists.[11]

3. Cause

Once the diagnosis of CKD is confirmed and the CKD stage is established, it is important to also consider the underlying cause and progression of CKD.[3] The leading causes of ESKD are shown in Figure 2. Family, medication and social histories are key as part of this assessment. Myeloma should be excluded in older adults or anyone with other suggestive features (e.g. anaemia, hypercalcaemia, infections, bone pain) as urgent referral to haematology may allow disease-specific therapy.

If the cause of CKD is uncertain, that may be reason to refer to nephrology, and a renal biopsy may be considered if there is haematuria and/or proteinuria suggesting a glomerular disorder.[12] Some renal disorders require specific intervention, such as immunomodulatory therapy, whilst others benefit from more generic therapies (discussed below). Identifying the aetiology of CKD is also helpful for education/discussion with the patient and can help predict progression (though this is variable). Despite investigation, the cause of a patient's CKD remains unknown in around 15% of people reaching ESKD.[13] In this situation the focus continues on prevention of progression, identification of metabolic complications of renal disease and preparation for renal replacement therapy where appropriate.

4. Imaging

Ultrasound is the imaging modality of choice for the kidneys and can be extremely informative. Ultrasound can confirm the following:

1. Presence of two kidneys: a kidney may be congenitally absent, atrophied or been surgically removed.
2. Renal size: small kidneys on ultrasound suggest chronicity of renal disease while nephromegaly is associated with diabetes, polycystic kidney disease and infiltrative disorders.
3. Cortical thickness: cortical thinning is a sign of chronicity.
4. Obstruction of the renal tract: hydronephrosis ± hydroureter is suggestive of an underlying urological disorder and the level of the obstruction may be apparent on the scan.
5. Cysts: most commonly simple cysts are noted that are of no consequence, however, a complex cyst may be malignant and require urological assessment. Multiple, usually bilateral cysts in enlarged kidneys are suggestive of polycystic kidney disease.

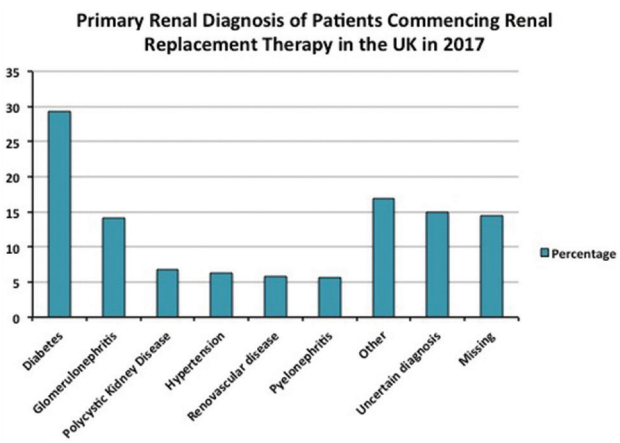
AKI in context of CKD

Patients with CKD are at higher risk of developing AKI – both in frequency and severity. They are also more likely to require renal replacement therapy for AKI and less likely to recover to their previous baseline level of renal function.[14] Therefore, it is essential to quickly identify any reversible causes of deterioration.

The causes of an acute deterioration in a patient with CKD remain the same as the causes of AKI in the general population and the following should be considered:

- Pre-renal: hypovolaemia (reduced oral intake, increased losses, e.g. fever, gastrointestinal fluid loss), reduced renal perfusion (reduced cardiac output, e.g. heart failure, myocardial infarction, arrhythmia, sepsis, renovascular disease, e.g. renal artery stenosis, thrombosis).
- Post-renal: commonly bladder outflow obstruction in older men (especially benign prostatic hypertrophy and prostatic carcinoma) though other obstructive causes should be considered, including stones, anticholinergic medication, neurological causes of incomplete bladder emptying.
- Renal: toxins [e.g. nonsteroidal anti-inflammatory drugs (NSAIDs), angiotensin-converting enzyme inhibitors/angiotensin-receptor blockers, aminoglycosides, contrast, rhabdomyolysis, haemolysis, hypercalcaemia], interstitial nephritis, glomerulonephritis, uncontrolled hypertension, urosepsis.

Figure 2 Leading causes of end-stage kidney disease in the UK with figures from the UK Renal Registry Report 2018[2]



If medications are discontinued owing to AKI it is essential that a medication review takes place in a timely fashion after discharge to plan the controlled reintroduction of medication, such as diuretics and renin-angiotensin system (RAS) blockade, to avoid the risk of readmission to hospital with decompensated cardiac failure.[15]

Who might benefit from referral to nephrology?

This requires a tailored approach and depends on the individual patient and circumstances. Specialist referral should be *considered* in patients with the following:[5]

- CKD of unclear cause.
- CKD stage 4 for at least an initial assessment and input. In some patients, if their CKD remains stable and uncomplicated, management may subsequently be with primary care.
- Proteinuria (ACR >70 mg/mmol) unless they are known to have diabetes and already receiving appropriate therapy.
- Rapidly progressive CKD, regardless of the stage: 25% reduction in eGFR over 12 months or sustained reduction in eGFR 15% per year for >12 months.
- Hypertension that is difficult to control despite four agents at appropriate doses.
- Suspected renal artery stenosis.
- Known or suspected rare/genetic causes of CKD.
- Problematic symptoms or complications related to their CKD.

A graph of kidney function over time can be extremely helpful to identify those with progressive disease. Graphs can be generated in the laboratory and reviewed to flag those with progressive decline to the general practitioner for consideration of referral.[16]

Part 2: management of CKD

Assessment of progression of CKD

The progression of CKD is often variable and nonlinear, though the pattern of change in preceding weeks, months and years can help predict future progression. Patients can vary between having very stable CKD for many years to relatively rapid progression, and although this is sometimes difficult to predict, there are certain risk factors that make it more likely that CKD will progress.

For CKD to be described as progressive, there should be three eGFR or creatinine tests over a period of >90 days that show declining renal function. It is often difficult to appreciate whether CKD is stable or slowly (or rapidly) progressing and plotting a graph of eGFR or creatinine can be most informative – both as medical professionals and for discussion with the patient.[16]

The risk factors for progressive CKD are:

- proteinuria
- hypertension
- pre-existent CKD (and specific cause, e.g. adult polycystic kidney disease)
- AKI (increased subsequent risk even if function returns to baseline)
- diabetes
- smoking
- cardiovascular disease
- African/Asian ethnicity
- chronic NSAID use
- untreated urinary outflow tract obstruction.

Accelerated progression of CKD is defined as a 25% reduction in GFR over 12 months or a sustained decrease of 15% per year, and these patients should be referred to nephrology for assessment as these groups of patients are at risk of further progression and development of ESKD.[5] There are prediction equations now available online (Kidney Failure Risk Equation and KDIGO equation) that are based on variables, such as age, sex, eGFR and proteinuria, and can help predict progression to ESKD.[17,18] These have been proven to be more accurate than eGFR alone. The KDIGO risk calculator also includes information about cardiovascular risk.[18]

The frequency of monitoring of blood tests should be intensified in patients with more advanced disease and/or heavy proteinuria in order to detect patients with progressive CKD or an acute deterioration requiring intervention. Table 2 suggests frequency of monitoring as outlined in the NICE guidance; however, this should be modified according to the individual's risk of progression and agreed with the patient.[5]

Medical management of CKD

General approaches

As with most long-term conditions, engaging patients in the diagnosis and management of their condition is encouraged and may improve outcomes. There are a number of modifiable lifestyle factors where interventions may delay progression of CKD and reduce both mortality and morbidity. Tailoring the advice to the individual patient can help with engagement;

CKD stage	ACR <3 mg/mmol	ACR 3–30 mg/mmol	ACR >30 mg/mmol
Stage 1 (eGFR ≥ 90 ml/min/1.73 m^2)	≤1	1	≥1
Stage 2 (eGFR 60–89 ml/min/1.73 m^2)	≤1	1	≥1
Stage 3A (eGFR 45–59 ml/min/1.73 m^2)	1	1	2
Stage 3B (eGFR 30–44 ml/min/1.73 m^2)	≤2	2	≥2
Stage 4 (eGFR 15–29 ml/min/1.73 m^2)	2	2	3
Stage 5 (eGFR <15 ml/min/1.73 m^2)	4	≥4	≥4

Table 2 Recommended frequency of monitoring of eGFR (number of times per year) by eGFR and ACR

ACR: albumin:creatinine ratio; CKD: chronic kidney disease; eGFR: estimated glomerular filtration rate

working together as part of a multiprofessional team with the person at the centre is key.[19]

The major goals of therapy vary depending on the severity of CKD:

- CKD stage 3 – cardiovascular risk reduction and prevention of progression of CKD.
- CKD stage 4 – consideration of referral to nephrologist and management of metabolic complications of CKD.
- CKD stage 5 – shared decision-making regarding renal replacement therapy vs conservative kidney management and preparation for dialysis/transplantation (as appropriate).

General advice regarding a healthy active lifestyle, maintaining a healthy weight, avoiding recreational drugs and engaging in regular exercise are applicable to most, at all stages of CKD.

Specific approaches

- **Hypertension:** We should aim to optimise BP control, as per KDIGO international hypertension guidelines, for CKD patients, though this should be individualised based on age, comorbidities and risk of progression of CKD. In patients with CKD with proteinuria regardless of aetiology (ACR >30 mg/mmol) we should aim for a BP target <130/80 mmHg, otherwise the target should be <140/90 mmHg.[20] However, these strict targets can be very hard to achieve in clinical practice. From the SPRINT trial, there is some evidence that more aggressive BP targets (systolic BP <120 mmHg) improve outcome and reduce cardiovascular mortality and morbidity.[21] This study excluded several relevant groups of patients including people with diabetes, a history of stroke, recent cardiac event, polycystic kidney disease or symptomatic heart failure. The study also used stringent methods of measurement including an average of three BPs taken during a period of rest, which is not comparable to the casual BP measurements often taken in the hospital clinic setting.[22] There were significant rates of adverse events including AKI in the lower BP target group. At present, this has not altered the national guidance in the UK.
- **Proteinuria and RAS blockade:** RAS inhibition with ACE inhibitors (ACEi) or ARBs remains the mainstay of treatment for people with CKD and proteinuria (ACR >30 mg/mmol) and they should be the first-line agent for people with diabetes or younger patients with hypertension. RAS blockade reduces BP and proteinuria, slows progression of CKD and has a beneficial effect on cardiovascular disease and mortality. However, ACEi/ARBs can be a double-edged sword as they may be harmful in the minority with pre-existing low renal blood flow, such as bilateral renal artery stenosis.
- **Difficulties with ACEi/ARBs:**
  - Potassium >6.5 mmol/l: repeat urgently ± initiate emergency management, stop ACEi and monitor closely until <6 mmol/l.
  - Potassium 6–6.5 mmol/l: repeat to ensure result not spurious, identify/stop concurrent medication that causes hyperkalaemia and offer dietary low potassium advice (this can be arranged by the nephrology team if requested). If potassium is still >6 mmol/l on repeat testing then reduce or stop ACEi/ARB.[23] Dietary intervention with low potassium advice may allow continuation of treatment, or a sufficient fall in serum potassium to restart.
  - Reduction in eGFR/increase in creatinine:
    - If creatinine increase is <20%: continue and recheck in 2 weeks.
    - If creatinine increase >20%: investigate for other causes of deterioration: reduction in blood volume/concurrent use of NSAIDs (must be discontinued).
    - If no other cause found, reduce dose or stop and repeat serum creatinine.[5]
  - Dual blockade with ACEi and ARBs is no longer recommended owing to increased risk of adverse events.[24]
  - 'Sick day rules'. For some patients with an intercurrent illness, particularly with hypovolaemia, withholding ACEi/ARB may be beneficial, but there is concern that introducing routine 'sick day rules' may cause unintended harm owing to reluctance to restart these

medications and loss of the prognostic benefit. To date, there have been no clinical trials to support this guidance.[25]

- **Cardiorenal syndrome:** The coexistence of renal dysfunction and chronic heart failure is extremely common and management can be challenging. A full discussion of this is outwith the scope of this paper, however, very helpful and pragmatic advice can be found in the consensus guidelines from the Renal Association and British Society for Heart Failure.[26]
- **Glycaemic control:** The duration and severity of hyperglycaemia is directly linked to end-organ damage (including the development of diabetic kidney disease), particularly in type 1 diabetes.[27,28] Optimising glycaemic control can delay progression of CKD. That being said, too stringent control also is associated with adverse outcomes. The 2019 CREDENCE trial demonstrates that treatment with the sodium-glucose 2 cotransporter inhibitor canagliflozin for patients with type 2 diabetes and albuminuric CKD stage 1–3 is associated with a reduced risk of ESKD, reduced progression of CKD and a lower risk of cardiovascular events.[29] However, the current license for use in the UK requires eGFR >60 ml/min/1.73 m^2 at initiation and therapy must be discontinued if eGFR drops <45 ml/min/1.73 m^2 though this may change in view of the latest trial evidence. However, there have been some safety concerns raised that require further investigation. Trials are ongoing to assess the efficacy in nondiabetic kidney disease.
- **Smoking:** The cardiovascular effects of smoking with sympathetic activation from nicotine and subsequent hypertension are well documented and this can also affect the renal vasculature and blood flow. As well as this, patients who smoke have higher rates of progression of proteinuria and renal decline.[30] Encouraging smoking cessation remains a key focus for these patients in optimising management of their CKD and reducing their cardiovascular risk.
- **Diet:** If the patient has hypertension or oedema, then a diet avoiding additional salt can be considered but the evidence in CKD patients, particularly patients with diabetes, is inconclusive and indeed very low salt intake may cause harm.[31] Low potassium dietary advice for those with CKD and hyperkalaemia, however, can make a significant difference. Advice about fluid intake will vary depending on urine output/fluid status/serum albumin and should be tailored to the individual patient, but for the majority, avoiding hypovolaemia is all that is required.[32,33] Specialist renal dietitians have a key role in the multiprofessional team management of CKD patients.
- **Urinary outflow obstruction:** This should be addressed either with medication, catheterisation/nephrostomy or urology referral and input, depending on the site of obstruction. Delaying this will lead to potentially avoidable renal decline.
- **Drug dosing in CKD:** Many medications are renally excreted and therefore require dose reduction for people with CKD. A detailed review of this is outwith the scope of this paper. However, we recommend consulting a specialised up-to-date resource that advises on dose adjustment to ensure safe administration of medication for people with CKD, such as the Renal Drug Handbook, which is used extensively in the UK.[34]

Management of complications of CKD

These are primarily managed by the nephrology multiprofessional team. Metabolic complications are unlikely to arise until the person develops stage 4 CKD.

- Renal anaemia: Patients should be iron replete before receiving erythropoietin, and often require intravenous iron to achieve this. Causes of iron deficiency should be considered, such as gastrointestinal malignancy/blood loss. Target haemoglobin for people with CKD receiving erythropoetin is 100–120 g/l.[35]
- Metabolic acidosis: This is common in CKD stage 4/5 and is associated with poor nutritional status, muscle wasting and loss of bone density, and may contribute to progression of renal disease.[36] Aim to correct this using oral sodium bicarbonate in people with CKD stage 4/5 and a serum bicarbonate of <20 mmol/l.[5]
- CKD-related mineral bone disease:
  - *Hypocalcaemia:* Vitamin D deficiency should be identified and corrected first. However loss of hydroxylation of vitamin D by the kidney subsequently leads to hypocalcaemia in more advanced CKD. People with CKD stage 4/5 and ongoing issues should be started on a vitamin D analogue (alfacalcidol or calcitriol) to correct this. Calcium must be monitored.[5]
  - *Hyperphosphataemia:* Phosphate is usually excreted by the kidneys and reduction leads to hyperphosphataemia. Patients may require a phosphate binder with their meals to reduce absorption of dietary phosphate. Dietary advice to reduce their phosphate intake is also helpful.
  - *Hyperparathyroidism:* As a response to hypocalcaemia, patients often develop secondary hyperparathyroidism. Vitamin D replacement is usually effective in the first instance but some patients go on to require further medical management or parathyroidectomy.
- Hyperkalaemia: This is common and may be troublesome. Avoiding culprit medication and addressing dietary factors are first-line therapy. Correction of chronic metabolic acidosis can help, as can diuretics to promote kaliuresis for some patients.
- Gout: This frequently seen in people with CKD and reduced dose colchicine is the preferred treatment, rather than NSAIDs or steroids. They frequently require long-term urate lowering therapy to prevent recurrent attacks. Treatment of asymptomatic hyperuricaemia is not currently recommended.

Useful tips for management of CKD

Lastly, here are the answers to the most commonly asked questions regarding the management of people with CKD.

- Trimethoprim inhibits tubular secretion creatinine and so falsely causes a decrease in *estimated* GFR without

affecting the true GFR. This effect is more pronounced in CKD patients (rise of up to 20–50%) and can take as long as 14 days to resolve. Trimethoprim also reduces excretion of potassium leading to hyperkalaemia, particularly if given with other medication that can cause hyperkalaemia.

- Gentamicin can be administered safely in CKD but correct monitoring and dosing are of paramount importance. AKI associated with aminoglycosides has been considered to be due to a cumulative effect of high trough doses, however, an association with single doses of gentamicin and AKI has been shown in a large study using gentamicin as prophylaxis in orthopaedic operations.[37]
- Owing to the effect on renal vasculature and perfusion, all NSAIDs can cause a fall in GFR and hyperkalaemia and should be used with caution in CKD. Renal function, BP and fluid status should be regularly assessed if they are deemed the most appropriate treatment.
- Metformin does not cause CKD but the drug clearance is reduced in CKD and current guidance advises stopping metformin when creatinine clearance <30 ml/min/1.73 m^2.
- Reduced cardiac output with fluid overload associated with heart failure reduces renal perfusion causing a secondary effect of reducing GFR. In these patients diuretic therapy may *improve* renal function by reducing renal venous pressure.[26]
- Be mindful that a patient with advanced CKD may require fistula formation and avoid repeated venepuncture or cannulation of the forearm veins (the dorsum of the hand is the preferred site).
- Bladder outflow obstruction is common, especially in the elderly male population. This should be excluded in the first instance in this population with an acute deterioration in eGFR. Similarly in patients with a history of stone disease, malignancy or previous obstruction, urology may be the most appropriate specialty in the first instance.

Conclusion

CKD is very common; people with CKD often have a wide range of comorbidities and, therefore, many other specialists will be involved in their care. There are several areas where we can intervene to delay progression of CKD and reduce the risk of adverse outcomes, therefore, early recognition and appropriate specialist referral are essential. Involving the individual in their management with a holistic, tailored approach is key in order to provide the best possible person-centred care.

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