

Chronic kidney disease frequently asked questions

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Introduction

NHS Employers and the General Practitioners Committee (GPC) of the British Medical Association (BMA) have published these frequently asked questions (FAQs) in response to queries received from primary care organisations and practices, in relation to the diagnosis and management of chronic kidney disease following its inclusion in the Quality and Outcomes Framework (QOF) in 2006. These FAQs have been updated in autumn 2009.

About eGFR

1. What is eGFR?

eGFR (estimated Glomerular Filtration Rate) is a measure of renal function. It is a more sensitive indicator of renal impairment than serum creatinine alone. Patients can have significant renal impairment even with a serum creatinine in the normal range. It is possible to lose up to 50 per cent of renal function before the creatinine becomes elevated, especially in the elderly.

2. Why has eGFR been adopted?

The use of eGFR follows recommendations in the Renal National Service Framework (NSF).¹ It is a more sensitive marker of kidney dysfunction than serum creatinine alone and it will allow earlier identification of patients developing chronic kidney disease (CKD). This is important as these patients have an increased cardiovascular risk compared with the general population and may benefit from risk factor modification. The use of eGFR will also facilitate identification of patients with more advanced CKD previously not recognised as such (for example an 80 year old with a creatinine of 160).

3. How is eGFR calculated?

eGFR is calculated from the serum creatinine, age, sex and ethnicity of the patient, using a formula called the modification of diet in renal disease (MDRD) formula. Most chemical pathology laboratories are now reporting eGFR automatically alongside any request for creatinine. For the aficionado, an online calculator is available via the internet at: www.renal.org

However, there are issues (see **Q4**) around standardisation of creatinine assays between different laboratories, so priority should always be given to the laboratory reported eGFR over an inhouse GFR calculated online.

Many labs have opted to report the numerical value for eGFR only if the eGFR is 60 or under. If eGFR is over 60 they will simply report it as > 60mL/min/1.73m².

¹www.dh.gov.uk/en/Publicationsandstatistics/Publications/PublicationsPolicyAndGuidance/DH_4101902

4. Are there inter-laboratory differences in eGFR calculation and reporting?

Yes. There are two issues around laboratory reporting.

There is variation in lab reported eGFR. There are different assays for the measurement of serum creatinine concentration in the UK. Therefore the creatinine in one laboratory is not necessarily equivalent to that in another. However, efforts are in hand to improve things.

Technical detail: Since 2007 there has been a process to standardise creatinine assays against the gold standard ('IDMS' – Isotope Dilution Mass Spectrometry) method through the National External Quality Assurance Scheme (NEQAS). A specific IDMS version of the MDRD formula can then be applied; this version of the MDRD formula has a different constant to that in the eGFR calculators available online. This standardisation enables direct comparison of creatinine and eGFR values between different laboratories. Some laboratories do not yet produce standardised creatinine concentrations; in these cases, assay-specific versions of the MDRD formula (with different slope and intercepts) are applied to local (non-standardised) creatinine values to produce standardised eGFR results. As a result of the complexities, clinicians should therefore always use the lab calculated eGFR ahead of one they might derive in-practice using an online calculator.

Higher values of eGFR are less accurate. There are differences in the upper limit of eGFR reported as an exact value. In many laboratories, a numeric eGFR is reported up to 59ml/min/1.73m²; while others report GFR up to 89ml/min/1.73m², with higher readings reported as ≥ 90 ml/min/1.73m². It should be emphasised that estimates of eGFR become less accurate as true GFR increases. Changes in serum creatinine concentration within the normal range can be of importance. NICE (National Institute of Clinical Excellence) recommend that where the eGFR is reported as ≥ 60 ml/min/1.73m², a rise in serum creatinine of > 20 per cent should be regarded as significant.

Changes in eGFR within an individual patient are a highly reliable way of tracking changes in kidney function, though there may be some fluctuation between measurements especially if there has been a large protein containing meal pre-test or there has been undue delay in analysing the specimen. Looking at all previous serum creatinine measurements, not just the last two or three, is important to get an idea of trend over time.

5. Why is ethnicity in the eGFR formula?

Afro-Caribbean people tend to have proportionally greater muscle mass than non Afro-Caribbean and therefore produce more creatinine. Consequently a creatinine of, for example, 150 in an Afro-Caribbean represents better renal function than the same creatinine value in a non Afro-Caribbean patient. NICE recommends applying a correction factor of 1.21 in all people recorded as

having black or Afro-Caribbean ethnicity. The laboratory will however generally not know the ethnicity of the patient. For that reason the eGFR will always be calculated for a non-Afro-Caribbean patient, with instructions on the report to multiply by 1.21 for an Afro-Caribbean patient. As ethnicity recording is patchy it is sensible to apply this correction to all black people.

There is no adjustment currently recommended for Asian or mixed race populations.

6. Will eGFR replace serum creatinine?

No. Creatinine will continue to be reported, but will be supplemented by eGFR.

7. What is the difference between creatinine clearance and eGFR?

Measurement of creatinine clearance gives an estimate of the actual glomerular filtration rate (GFR), as does eGFR. However, eGFR is less cumbersome than performing creatinine clearance, which requires a 24 hour urine collection.

8. What is the normal range for eGFR?

An eGFR $> 90\text{mL}/\text{min}/1.73\text{m}^2$ is considered normal in a young fit adult (although few labs report numeric values of eGFR above $60\text{mL}/\text{min}/1.73\text{m}^2$ – see Q4.ii). However, eGFR frequently declines with age, on average by between 6 and $9\text{mL}/\text{min}/1.73\text{m}^2$ per decade. Around half of females over 75 years and men over 85 years will have an eGFR under $60\text{mL}/\text{min}/1.73\text{m}^2$. It is believed that declining eGFR with increasing age arises in the context of vascular co-morbidities which are common in the elderly, rather than as a consequence of the ageing process per se.

An eGFR of 60 to $90\text{mL}/\text{min}$ does not in itself indicate CKD – for a formal diagnosis of CKD additional markers of damage are required as well. These markers can either be on imaging (for example, polycystic kidneys) or abnormal urine findings (for example, microalbuminuria, proteinuria or microscopic haematuria). The finding of an eGFR of 60 to $90\text{mL}/\text{min}/1.73\text{m}^2$ should not in itself prompt investigations looking for these markers, unless there are other signs of potential renal pathology such as haematuria.

An eGFR of $< 60\text{mL}/\text{min}$ present on at least two occasions less than three months apart, however, does represent CKD.

9. Does eGFR change with age?

Yes. Beyond the age of 40 a progressive loss of eGFR of up to $1\text{mL}/\text{min}/\text{year}$ is commonly seen, particularly amongst patients with hypertension or vascular disease. Whether this should be considered 'normal' is controversial.

Although eGFR can decline with increasing age, this in itself should not preclude these patients from receiving care as outlined by the QOF indicators. This care seeks to protect these patients from experiencing preventable cardiovascular complications to which they are more vulnerable. It is not possible to give an age cut off after which CKD should not be diagnosed or hypertension treated. The decision to treat CKD should be made in conjunction with the patient and where

appropriate their carer, taking into account issues such as their general health and quality of life.

The eGFR calculation is not valid in children (those under 16 years). The Schwartz formula is valid for use in this age group. However, should kidney disease be suspected in a child, this would in itself be a reason for referral.

10. Is eGFR useful in acute renal failure?

No. However, if an unexpected eGFR < 60 ml/min/1.73m² is reported and there are no previous eGFR readings available for comparison it is worth looking at historical serum creatinine values. If there has been a sudden rise in serum creatinine or there are no historical values available for comparison, then a repeat creatinine and eGFR should be requested urgently (within two weeks) to exclude any sudden increase in creatinine which may represent acute renal failure.

11. What should practices do if they do not have access to eGFR?

Practices should all have access to eGFR by now. In the rare circumstances in which they don't, practices should use one of the available calculators. Two suggested strategies (which can be used alone or in combination) are:

- Calculate eGFR for each serum creatinine result that comes into the practice. This can be done using an online calculator available at www.renal.org
- Export age, gender and creatinine from your GP computer system into a spreadsheet which will run the calculation for you. This can be downloaded from www.pcel.info/gfr/ (N.B. You must 'enable macros' if asked to do so when you download this; full instructions are available via a tab at the bottom of the spreadsheet). Once in the spreadsheet the data can be sorted by stage of CKD. We would recommend that you flag patients for a blood pressure review at their next visit.

Some of the software manufacturers have developed options which allow eGFR to be estimated for the whole practice population. Practices should contact their computer system supplier about this. However, do remember that there are inter-laboratory differences in creatinine assays (see **Q4**) and that where the inhouse calculated eGFR differs from the laboratory eGFR, priority should be given to the laboratory result.

There is no easy way of defining a serum creatinine cut-off as an alternative to using eGFR. This is because creatinine is a relatively poor measure of renal function – it is possible to lose up to 50 per cent of renal excretory function before the serum creatinine concentration rises above the upper limit of normal.

12. What are GPs expected to do when they find someone with a low eGFR?

- First compare with previous eGFR to see if there has been a significant change in renal function. If there are no previous eGFR available for

comparison then compare current creatinine with historical creatinine readings to assess stability.

- If there are no historical eGFR or creatinine readings available for comparison, or if there has been a sudden change in renal function, then consider acute kidney injury (AKI, previously called acute renal failure). The eGFR should be repeated within two weeks and urine protein quantified (see **Q28**). Consider need for urgent referral, which, depending upon the severity of AKI and the patient's clinical condition may be necessary before the eGFR is repeated.
- If the patient is acutely unwell consider AKI and repeat the test immediately or discuss/ refer for a specialist opinion.
- Check if the patient is on the CKD register. If the patient has two eGFR readings less than 60ml/min/1.73m² over three months apart (with no readings of \geq 60ml/min/1.73m²- in between) then a diagnosis of Stage 3–5 CKD should be made and the patient added to the CKD register (see **Q44**).

The patient should be managed according to the principles set out below.

If eGFR is low:

- The first priority is to check the patient's blood pressure. The target for blood pressure should be 120-139/<90. However, if there is proteinuria (ACR \geq 70mg/mmol, equivalent to PCR \geq 100mg/mmol) a lower target of 120-129/<80 is recommended. We recognise that the QOF target has been set at 140/85 because of the very real difficulty in controlling blood pressure in many of these patients. The best way to get most patient's blood pressure below the audit threshold is to aim for the 'target' of 120-139/<90. However, the evidence base supports the use of the 120-139/<90 target (or 120-129/<80 in people with diabetes and/or proteinuria).
- In people with diabetes and/or significant proteinuria (ACR \geq 30mg/mmol, equivalent to PCR \geq 50 mg/mmol) treat with an ACE-I or ARB and other agents to reduce blood pressure measurements to target.
- The two proteinuria threshold are:
 - ACR (albumin:creatinine ratio) \geq 70mg/mmol, equivalent to PCR (protein:creatinine ratio) \geq 100mg/mmol, which alters the blood pressure target from 120–139/<90 to 120–129/<80
 - ACR \geq 30mg/mmol, equivalent to PCR \geq 50 mg/mmol, which is the threshold blood pressure for using an ACE-I or ARB.
- Check potassium before starting and recheck along with creatinine after two weeks of starting, after changes in dose or if any severe intercurrent illness. ACE-I (Angiotensin I-converting enzyme) or ARBs (angiotensin receptor blockers) are the best drugs to treat hypertension in proteinuric CKD although they can sometimes reduce renal perfusion. These agents are often used in non-proteinuric patients to achieve the target blood pressure as part of combination therapy. It is not justified to change patients who have good blood pressure control on other agents and no proteinuria to an ACEi/ARB just because of CKD Stage 3.

NICE recommend renal referral where blood pressure is uncontrolled despite use of four or more anti-hypertensive agents.

- Next test for proteinuria and check the patient is not anaemic for stage 3b disease and below. Anaemia is generally defined in CKD as Hb<11g/dL. (See **Q23** 'how anaemia is defined in CKD').
- Manage other cardiovascular risk by controlling cholesterol and encouraging smokers to quit.
- Improve control of heart failure and diabetes. Conduct a medication review for drugs which impair renal function. In men consider whether prostatic disease may be causing outflow problems.

Please see other relevant other sections:

- How CKD is classified (see **Q15**)
- How often to re-check creatinine (see **Q36**)

Please also see referral guidance (see **Q37–42**).

13. Does eGFR vary and what accounts for this variability?

Yes. The analytical and biological variability of eGFR is estimated to be approximately 5 per cent. Important sources of variation include:

- Ingestion of cooked meat. Creatinine is largely a product of protein breakdown and therefore ingestion of high protein meals (largely meat) can change serum creatinine concentration and therefore eGFR. Ingestion of cooked meat may cause a rise in creatinine of 30–40µmol/L or more at four hours, returning to baseline by 12 hours.
- Change in muscle mass. Loss of muscle mass will result in reduced generation of creatinine and lower eGFR for the same 'actual' renal function. In exceptional cases where muscle mass is greatly reduced expert assistance may be required to interpret eGFR – especially in someone with other vascular disease, e.g. a patient with diabetes and amputation.
- State of hydration.
- Changes in renal perfusion. This is particularly evident in patients with heart failure in whom small changes in, for example, doses of diuretics and angiotensin converting-enzyme inhibitors, can cause wide fluctuations in eGFR.
- Intercurrent illness – especially urinary tract infection.
- Delay in centrifugation of blood samples can cause an increase in creatinine concentration.

About chronic kidney disease

14. What is chronic kidney disease?

Chronic kidney disease (CKD) is the current term which has replaced the terms chronic renal failure and renal impairment. It is characterised by one or both of: a reduction in kidney excretory function, and the presence of other markers of kidney damage (for example, protein in the urine). The prevalence of CKD increases with age and it is more common in females than in males. The condition is classified into stages of severity on the basis of the eGFR; the less severe stages are extremely common.

Stage	GFR (ml/min/ 1.73m ²)	Percentage population (QICKd study data)	Description
No serum creatinine measure		64.2%	We cannot estimate renal function for people who have not had a creatinine measured. However, these are mainly young people and around 75 per cent to 80 per cent of people over 60 years have a serum creatinine recorded.
1	≥ 90	23.0%	Normal or increased GFR, with other evidence of kidney damage
2	60 – 89	7.3%	Slight decrease in GFR, with other evidence of kidney damage
3a	45 – 59	4.5%	Moderate decrease in GFR, with or without other evidence of kidney damage
3b	30 – 44	0.7%	
4	15 – 29	0.1%	Severe decrease in GFR, with or without other evidence of kidney damage
5	< 15	0.0%	Established renal failure

Table 1: Stages of CKD, and the proportion of the population with no creatinine measure and in each stage of CKD (QICKd study data)

Practical tip: To reduce variability perform eGFR tests under standard conditions where possible, e.g. after an overnight fast but taking water. This can be combined with measurement of fasting lipids and blood glucose in patients with vascular co-morbidities, and will usually avoid long delays in despatch of the blood sample, so it is processed by the laboratory within 12 hours of venepuncture.

Inter-laboratory variation in eGFR has been minimised by the standardisation GFR calculations (see **Q4**).

The stages of Chronic Kidney Disease (CKD) are set out in Table 1. This table also gives the proportion of the population who don't have a serum creatinine measure and in each stage of CKD, as found in the QICKd study.

People with CKD can be further 'risk stratified' according to the presence or absence of protein in the urine (proteinuria). Significant proteinuria is associated with an increased risk of renal disease progression towards dialysis, and of cardiovascular disease (CVD). CKD is typically asymptomatic until the more advanced stages of disease.

Most people with CKD have renal function that is relatively stable over time, and will never reach dialysis dependence. However all are at risk of cardiovascular disease. Vascular co-morbidities (for example hypertension, diabetes, peripheral vascular disease and heart failure) are more common in CKD; it is also recognised that reduced eGFR and proteinuria are independent risk factors for cardiovascular disease. Blood pressure control is effective in reducing cardiovascular risk and slowing progression of CKD.

15. How is CKD classified?

CKD is divided into five stages based on eGFR, according to the Kidney Disease Outcomes Quality Initiative (K-DOQI) classification. The lower the eGFR, the worse the stage of CKD. A minimum of two eGFR values at least three months apart should be used to diagnose and classify CKD – temporary rises in creatinine are common and a diagnosis of CKD should be based on a sustained decline in renal function. People with an eGFR of 60mL/min/1.73m² or over should not be considered to have CKD unless there is other evidence of kidney damage.

In Stages 1 and 2 markers of kidney damage are required for a diagnosis of CKD. These markers can either be on imaging (for example, polycystic kidneys) or abnormal urine findings (for example, microalbuminuria, proteinuria or microscopic haematuria).

NICE recommend the addition of the suffix (p) to denote the presence of significant proteinuria (ACR \geq 30mg/mmol or PCR \geq 50mg/mmol) (see **Q29**). These patients should be treated with ACE-I/ARB (see **Q25**).

Stages of Chronic Kidney Disease and complications

16. Why has Stage 3 been subdivided into Stage 3a and b?

The subdivision of Stage 3 CKD into Stage 3a (eGFR 45 - 59ml/min/1.73m²) and Stage 3b (eGFR 30 - 44ml/min/1.73m²) has been adopted in the UK following a recommendation from NICE in their 2008 Guidance on CKD (guidance.nice.org.uk/CG73). Patients with Stage 3b disease are at far higher risk of cardiovascular disease and end-stage renal disease than those with Stage 3a disease, and should therefore be regarded as an important target group in

primary care. Stage 3b disease cannot be explained on the basis of the ageing process alone, as some will argue for early stage 3a disease.

17. What is the expected prevalence of CKD in a practice with a list size of 10,000 patients?

A practice of 10,000 will have at least 500 patients with Stage 3 to 5 CKD, with around 20 of these in Stage 4 and ten in stage 5. A more elderly and more deprived practice population are likely to have a higher prevalence. Of the 500 with Stage 3, 84 per cent (422) will have Stage 3a and 16 per cent (68) Stage 3b (QICKD trial data).²

About 90 per cent of people with Stage 3 to 5 CKD will have hypertension, 40 per cent will have vascular disease and 30 per cent will have diabetes.

Full details of the current prevalence of Stage 3 to 5 CKD is recorded on the NHS Information Authority website (www.ic.nhs.uk) or at NHS Comparators (www.connectingforhealth.nhs.uk/systemsandservices/sus/delivery/comparators).

18. What are the most important facts about chronic kidney disease for primary care clinicians?

- CKD is very common; around five to six per cent of the population have Stage 3 to 5 disease. It is more common in women than men, though as renal function deteriorates the proportion of men increases.
- Common causes include diabetes, vascular disease, and, in males, obstructive renal disease.
- The majority (> 80 per cent) of patients have stable CKD and are at far higher risk from cardiovascular disease than they are of ever requiring renal replacement therapy. People with CKD are roughly 20 times more likely to die from CVD than progress to end stage renal failure. The all causes mortality rate in CKD is 30 to 60 times higher than in the general population. The presence of proteinuria indicates a higher cardiovascular and progressive kidney disease risk.
- NICE define progressive CKD as: a decline in eGFR of $\geq 5\text{ml/min/1.73m}^2$ within one year based on at least three readings or $\geq 10\text{ml/min/1.73m}^2$ within five years (see Q21)
- The mainstays of management are to:
 - identify patients requiring nephrology referral (Q38)
 - assess stability of disease (by monitoring creatinine, typically every six to 12 months)
 - address cardiovascular risk

² de Lusignan S, Gallagher H, Chan T, Thomas N, van Vlymen J, Nation M, Jain N, Tahir A, du Bois E, Crinson I, Hague N, Reid F, Harris K. The QICKD study protocol: a cluster randomised trial to compare quality improvement interventions to lower systolic BP in chronic kidney disease (CKD) in primary care. *Implement Sci.* 2009;4(1):39.

- assess any functional consequences of disease, particularly renal anaemia in those with stage 3b disease and below
- avoid/reduce exposure to nephrotoxic or potentially nephrotoxic drugs.

Please see national guideline documents for more details, available at:

NICE Clinical Guideline 73

www.nice.org.uk/nicemedia/pdf/CG073NICEGuideline.pdf

Early identification and management of chronic kidney disease in adults in primary and secondary care:

www.nice.org.uk/Guidance/CG73/NiceGuidance/pdf/English

Or the *Quick reference guide* at:

guidance.nice.org.uk/CG73/QuickRefGuide/pdf/English

NICE Clinical Guideline in Welsh

CG73 Nodi a thrin problemau hirdymor gyda'r arennau (clefyd cronig yr arennau): deall canllawiau NICE (file format MS Word)

guidance.nice.org.uk/index.jsp?action=download&o=43225

The Renal Association

www.renal.org/JSCRenalDisease/JSCRenalDisease.html

SIGN Guideline No. 103

Diagnosis and management of chronic kidney disease.

www.sign.ac.uk/guidelines/fulltext/103/index.html

Northern Ireland NHS guidance

www.crestni.org.uk/publications/chronic%2Dkidney%2Ddisease.html

Brief guidance for GPs distributed by RCGP

Blades S, Burden R. *Introducing eGFR. Promoting good CKD management.* CKD Guidelines Development Committee:

www.renal.org/eGFR/resources/eGFRnatInfoLflt0406.pdf

Articles for GPs and practice nurses

Straightforward introduction to CKD management in primary care

www.ncbi.nlm.nih.gov/pmc/articles/PMC1934049/

A more detail review can be found at

www3.interscience.wiley.com/cgi-bin/fulltext/119815475/PDFSTART

19. What is the target blood pressure in CKD?

A target of 120–139/<90 mmHg is recommended. A lower threshold of 120–129/<80 mmHg is recommended for:

- people with CKD and diabetes
- people with non-diabetic CKD and proteinuria at a level of ACR \geq 70mg/mmol or PCR \geq 100mg/mmol (see **Q28** for definitions of proteinuria); note that this is higher than the proteinuria threshold in non-diabetic CKD at which ACEI/ARB are beneficial (see **Q20**).

20. How do I manage cardiovascular risk in people with CKD?

Both reduced eGFR and proteinuria are markers of cardiovascular risk which should be documented. The most important aspect of management is blood pressure control.

Other CKD-specific aspects of cardiovascular risk management include:

- appropriate use of ACEI and ARB (see **Q27–29**)
- testing for and treating renal anaemia where appropriate (Stage 3b and below)
- regular recall to reduce and recognise renal progression.

Although people with CKD are at high cardiovascular risk, and it is recognised that the Framingham tables underestimate cardiovascular risk in CKD, current advice is that the indications for statin therapy in primary prevention, and the use of statins and aspirin in secondary prevention, are the same as those in the general population.

21. The eGFR seems very variable. How do I assess the stability of CKD?

NICE (clinical guideline 73) have defined progressive CKD by a fall in eGFR of \geq 5ml/min/1.73m² within one year (based on at least three readings) or a fall of \geq 10ml/min/1.73m² within five years.

However, as discussed in **Q4**, there are other important causes of variation in eGFR which should be considered and can cause difficulty. In assessing stability we recommend examining as many eGFR and serum creatinine readings over as long a time as possible. You may need to make a professional judgment as to whether you are seeing progressive CKD or normal fluctuation in an individual. If in doubt contact your local renal unit for advice.

NICE recommend that, in assessing progressive CKD, attention is focused upon those in whom continuing decline in eGFR at the observed rate would lead to the need for renal replacement therapy during their natural lifetime.

22. What is the expected prevalence of CKD in a practice with a list size of 10,000 patients?

A practice of 10,000 will have at least 500 patients with Stage 3 to 5 CKD, with around 15 of these in Stage 4 and five in Stage 5. A more elderly and more deprived practice population are likely to have a higher prevalence.

About 90 per cent of people with Stage 3 to 5 CKD will have hypertension, 40 per cent will have vascular disease and 30 per cent will have diabetes.

People with CKD are roughly twenty times more likely to die from cardiovascular disease than progress to end stage renal failure. The all causes mortality rate in CKD is 30 to 60 times higher than in the general population.

23. What drugs can make CKD worse?

All patients with CKD should have a medication review. The advice of a current copy of the British National Formulary (BNF) should be followed. The priorities for this medication review should be:

- Stop unnecessary medication which may impair renal function. For example, many patients may be on non-steroidal anti-inflammatory drugs (NSAID) which should be discontinued.
- Angiotensin converting-enzyme inhibitors (ACE-I) and angiotensin II receptor blockers (ARB) are the best drugs to treat hypertension in proteinuric CKD, although they can sometimes reduce renal perfusion. (See Q25)
- Renal metabolism and excretion of drugs might be impaired. For example, patients on analgesics, certain B-blockers (including atenolol), digoxin and allopurinol may all need their dose reducing.
- In diabetes sulphonylureas may accumulate and therefore short acting drugs are preferred. Metformin should only be used under specialist advice when eGFR is below 30mL/min/1.73m² (Stage 4 and 5 CKD). When eGFR is between 30 and 60mL/min/1.73m² the risk/benefit ratio of metformin should be assessed on an individual basis.

24. Can Metformin be used in people with CKD?

Metformin is a useful drug for treating diabetes and can be safely used in CKD. However specialist advice is needed if eGFR is < 30mL/min/1.73m² (Stage 4 and 5 CKD). The concerns with metformin are:

- it is excreted via the kidneys
- it is associated with lactic acidosis. However, trials and reviews show that metformin is safe and improves outcomes³; it is argued by some that the lactic acidosis risk is possibly overstated and not metformin related.⁴

³ Holman RR, Paul SK, Bethel MA, Matthews DR, Neil HA. *10-year follow-up of intensive glucose control in type 2 diabetes*. N Engl J Med. 2008 Oct;359(15):1577-89.

⁴ Salpeter SR, Greyber E, Pasternak GA, Salpeter EE. *Risk of fatal and nonfatal lactic acidosis with metformin use in type 2 diabetes mellitus: systematic review and meta-analysis*. Arch Intern Med. 2003;163(21):2594-602.

In practical terms, it is reasonable for GPs to use metformin in people with Stage 3 disease $eGFR > 30\text{mL}/\text{min}/1.73\text{ m}^2$. However, dosage reduction and specialist involvement should be considered as renal function declines towards this level through normal diabetes shared care arrangements. Practitioners should be more cautious about increasing metformin doses as renal function declines. The risks of lactic acidosis in people with $eGFR < 30$ are said to be related to the age of the patient and the dose of metformin: the higher the dose and the older the patient, the greater the risk.

25. When should Angiotensin Converting Enzyme Inhibitors ACE-I and Angiotensin Receptor blockers be used OR not used in CKD?

ACE-I and ARBs are the best drugs to use to control blood pressure in proteinuric CKD. They give the best outcome for patients. They are indicated in three broad settings in CKD:

- people with hypertension and non-diabetic CKD who have significant proteinuria ($ACR \geq 30\text{mg}/\text{mmol}$ or $TPCR \geq 50\text{mg}/\text{mmol}$)
- people with non-diabetic CKD and higher levels of proteinuria ($ACR \geq 70\text{mg}/\text{mmol}$ or $TPCR \geq 100\text{mg}/\text{mmol}$) regardless of level of blood pressure
- people with diabetes and microalbuminuria ($ACR \geq 2.5\text{mg}/\text{mmol}$ in men or $\geq 3.5\text{mg}/\text{mmol}$ in women) regardless of level of blood pressure.

Some small decline in $eGFR$ is expected as part of the effect of these drugs. However, if this decline is less than 25 per cent or progressive then discontinuation and specialist advice may be appropriate. This can be seen with, for example, hypovolaemia and with renal artery stenosis. It is therefore important to check electrolytes two weeks after starting, after increasing dose and during any intercurrent illness likely to cause hypovolaemia (including acute sepsis).

ACE-I/ARB are often used in non-proteinuric patients to achieve the target blood pressure as part of combination therapy. It is not justified to change patients who have good blood pressure control on other agents to an ACE-I or an ARB just because of non-proteinuric CKD Stage 3.

26. What attitude does the insurance industry take to CKD?

A risk assessment will be carried out for life, critical illness and income protection policies. Those at highest risk (for example, those who are young or have more advanced disease) are likely to be asked to pay increased premiums or excluded from cover if the risk is thought to be too high. However, although the absolute risk of cardiovascular disease is higher in older people with Stage 3 CKD, the increased risk relative to their peers without CKD may not be high enough to warrant higher premiums.

There is little formal risk assessment with travel insurance policies. Pre-existing or associated medical conditions are generally excluded. If comprehensive cover is required then patients should be advised to shop around to ensure they have an appropriate level of cover. Specialist insurers do offer travel policies for the

elderly or those with pre-existing conditions, but there is no guarantee that cover will be complete.

Testing for proteinuria and investigation of people with CKD

27. Who should have their urine tested for protein?

Everyone who is known to have CKD should be assessed for proteinuria annually using a quantitative test (ACR or, if ACR is not available, PCR). Patients with diabetes, who are having annual urinary albumin-creatinine ratio (ACR) tests as part of their screening for complications process, don't need to be additionally checked.

People at risk of CKD (particularly those with hypertension and vascular disease) should have a one-off quantitative proteinuria test. This does not need to be repeated on an ongoing basis unless there is significant proteinuria (ACR \geq 30mg/mmol or PCR \geq 50mg/mol). See **Q29** for values to be used in diabetes.

Testing of proteinuria is also indicated in other circumstances, for example, oedema or systemic disease.

Send an early morning urine for ACR or PCR and retest serum creatinine to obtain a further estimate of eGFR. (Strictly NICE only recommend an early morning urine where ACR is 30 to 70mg/mmol; however a routine of getting morning ACR measures performed may reduce the need for repeat tests). If the ACR is \geq 70mg/mmol (PCR \geq 100mg/mmol) or there is microscopic haematuria and ACR \geq 30mg/mmol (PCR \geq 50mg/mmol) renal referral is indicated. In asymptomatic individuals there is no evidence that urinary tract infection causes proteinuria; consequently there is no need to send the urine samples of asymptomatic individuals for microscopy and culture.⁵

28. How should I measure proteinuria?

NICE recommend that to detect and identify proteinuria reagent strips should not be used (unless they are capable of specifically measuring albumin at low concentrations and expressing the result as an albumin-creatinine ratio (ACR)). 24 hour urine collections for protein are similarly unnecessary.

Two quantitative tests to measure proteinuria from a random urine sample are available, the albumin:creatinine ratio (ACR) and the total protein:creatinine ratio (PCR). Both are sent in a plain white-topped urine bottle, ideally (but not essentially) on an early morning sample. The ACR test is the same as that already in use to measure proteinuria in people with diabetes.

NICE recommend that the ACR should be used for the early detection of proteinuria as it has higher sensitivity than the PCR. PCR is an alternative for

⁵ Carter JL. Tomson CR. Stevens PE. Lamb EJ. Does urinary tract infection cause proteinuria or microalbuminuria? *A systematic review. Nephrology Dialysis Transplantation.* 21(11):3031-7, 2006

monitoring proteinuria, particularly where ACR is not available. ACR remains the preferred test for people with diabetes.

ACR and PCR are both measured in mg (of urine protein) per mmol (of urine creatinine). Simply divide the value in mg/mmol by 100 to give the total daily 24 hour protein excretion in g/day. For example, if PCR = 100mg/mmol, then total daily protein excretion = 100 divided by 100 = 1 g/day. Urine creatinine is included to control for urine concentration, which is a major source of inaccuracy in reagent strips.

The ACR is the preferred measure of proteinuria and recommended by NICE. There are other proteins in urine so the PCR will always be greater than the ACR. The ACR is usually used to diagnose (and monitor) microalbuminuria, the first stage of diabetic nephropathy, and there is logic that a single test can be used to monitor the progression of CKD.

A repeat early morning sample to confirm the presence of proteinuria is indicated for intermediate values of ACR (30-70 mg/mmol) and PCR (50–100 mg/mmol).

29. What is the normal range for ACR (and PCR)?

Urine protein excretion is most simply categorised as one of: normal, microalbuminuria, and clinically significant proteinuria.

Microalbuminuria can only be assessed using an ACR test, as the PCR test is insensitive and inaccurate at low levels of proteinuria. Microalbuminuria is defined as an ACR of ≥ 2.5 mg/mmol in males and an ACR of ≥ 3.5 mg/mol in females. Microalbuminuria is of great significance in diabetes as an indicator of early diabetic nephropathy and of the need to prescribe ACE-I/ARB (see Q25).

Microalbuminuria is also a marker of kidney damage (in diabetics and non-diabetics), and the demonstration of microalbuminuria is sufficient for a diagnosis of Stage 1 or 2 CKD where the eGFR is > 60 ml/min/1.73m² (see Q15).

However, microalbuminuria is not currently an effect modifier for intervention (requiring ACE-I/ARB) in non-diabetic CKD, nor does it indicate the need for a suffix (p) in the CKD staging (see Q15).

Clinically significant proteinuria is defined by an ACR ≥ 30 mg/mmol or a PCR of ≥ 50 mg/mmol. It is these patients to whom the suffix (p) should be added to their staging, and who should receive ACE-I/ARB for non-diabetic CKD. People with higher levels of proteinuria benefit from tighter BP control (target 120–129/<80) and ACE-I/ARB regardless of level of blood pressure.

ACR mg/mmol	PCR mg/mmol	24 hour protein equivalent (g/day)	Significance in non diabetic CKD
30	50	0.5	Clinically significant proteinuria requiring ACE-I/ARB if hypertension present then add suffix (p) to staging and if BP target 120–139/<90 then consider renal referral if microscopic haematuria
70	100	1	If higher level proteinuria requiring ACE-I/ARB regardless of level of blood pressure then add suffix (p) to staging and if BP target 120–129/<80 then consider renal referral

Table 2: Quantitative proteinuria tests and their approximate equivalents

30. When and how should I test for haematuria?

When testing for haematuria use reagent strips rather than urine microscopy. There is no need to confirm the presence of haematuria using urine microscopy.

In CKD the urine should be tested for blood with a reagent strip if there is significant proteinuria on quantitative testing, i.e. ACR \geq 30mg/mmol or PCR \geq 50mg/mmol. In diabetic CKD, the urine should be tested for blood if microalbuminuria is present.

Therefore in a renal workup, quantitative urine protein measurement should precede urine stick testing. A result of 1+ should be regarded as significant.

The demonstration of microscopic haematuria with clinically significant proteinuria is an indication for nephrology referral. Persistent invisible haematuria (with or without proteinuria) should also always prompt the urological exclusion of urinary tract malignancy in the over 50s.

31. When should I test for anaemia in CKD?

Patients who are due to be referred, are symptomatic, or who have heart failure as a co-morbidity should have their haemoglobin checked.

Definitions of renal anaemia used in CKD differ but that used by NICE⁶, the European Best Practice Guidelines⁷ and KDOQI⁸ are recommended, i.e. using a threshold of 11g/dL to define anaemia and the need for treatment thereof.

People with Stage 3b CKD and below should be tested for anaemia. The mean haemoglobin falls, and the proportion with anaemia (Hb < 11g/dl) increases, as renal function declines in successive stages of CKD (see table 3). These data are

⁶ NICE Clinical Guideline 39: *Anaemia management in chronic kidney disease*. URL: <http://www.nice.org.uk/Guidance/CG39>

⁷ Locatelli F, Aljama P, Ba'ra'ny P et al. *Revised European best practice guidelines for the management of anaemia in patients with chronic renal failure*. *Nephrol Dial Transpl* 2004; 19(Suppl 2): 1–47.

⁸ KDOQI *Clinical practice guidelines and clinical practice recommendations for anemia in chronic kidney disease*. *Am J Kidney Dis* 2006; 47: S1–S145

from QICKD study practices in Southeast England,⁹ other areas with more cardiovascular co-morbidity may expect higher rates of anaemia.

	Population With eGFR & Hb recorded	Hb (g/dl)		People with anaemia (Hb <11g/dl)	
		n	Mean	Standard deviation	n
Mildly impaired renal function	35,669	14.1	1.4	489	1.6%
Stage 3a	6,956	13.6	1.4	223	3.5%
Stage 3b	1,140	12.8	1.6	127	12.0%
Stage 4	184	11.9	1.7	39	22.7%
Stage 5	32	11.5	1.9	10	37.0%

Table 3: Relationship between haemoglobin and anaemia in people with CKD

When considering if the anaemia is renal origin, practitioners should always consider whether there are any symptoms to suggest other causes, and examine the MCV and haematinics. Other causes should be sought if there are: suggestive symptoms; a micro- or macro-cytic picture; if the anaemia is disproportionate to the level of kidney function (anaemia is mild outside Stage 5 disease); or if there is a falling haemoglobin concentration with stable eGFR.

32. How is renal anaemia treated?

Patients with renal anaemia are treated with iron (frequently parenteral iron) and erythropoiesis stimulating agents (ESA)/erythropoietin (EPO). Advice should be sought from your local renal unit and/or from the excellent *IV iron in the community* document from the Anaemia Nurse Specialist Association (ANSA).¹⁰

33. Do I need to measure calcium, phosphate and parathyroid hormone in CKD?

There is no need to measure calcium, phosphate or parathyroid hormone (PTH) in patients with Stage 1–3b CKD. They should be measured in Stage 4 and 5 CKD. Whether they need monitoring on an ongoing basis is an individual patient decision. Advice should be sought from your local renal unit.

34. Do all CKD patients need a renal ultrasound?

No. Ultrasound should be reserved for those with progressive disease, advanced (Stage 4 or 5) disease, visible or persistent invisible haematuria, those over 20 years of age with a family history of polycystic kidney disease, or those with a palpable bladder/lower urinary tract symptoms.

⁹ de Lusignan S, Chan T, Gallagher H, van Vlymen J, Thomas N, Jain N, Tahir A, Nation M, Moore J, Reid F, Harris K, Hague N. *Chronic Kidney Disease management in southeast England: a preliminary cross-sectional report from the QICKD – Quality Improvement in Chronic Kidney Disease – study*. Primary Care Cardiology Journal 2009;12(1):33-9. Doi: 10.3132/pccj.2009.034 URL: <http://www.pccj.eu/download/3323>

¹⁰ Anaemia Nurse Specialist Association (ANSA). *A guide to the administration of intravenous iron for people with anaemia of chronic kidney disease (CKD) in a non acute hospital setting*. URL: http://www.anaemianurse.org/docs/CKD_Book_A4_Final_090408.pdf

Practices should refer to local care pathways prior to making a referral for a renal ultrasound.

See also: **Q40** - What information is required in a referral to a nephrologist?

35. Who should be tested for CKD?

eGFR should be measured annually in the following at-risk groups:

- hypertension
- diabetes mellitus
- cardiovascular disease (ischaemic heart disease, cerebrovascular disease, peripheral vascular disease, heart failure)
- patients receiving potentially nephrotoxic drugs (e.g. NAIDS, lithium, ciclosporin)
- structural renal tract disease, (prostatic hypertrophy, renal calculi)
- people with a family history of Stage 5 CKD
- people with a genetic risk of kidney disease
- multisystem disease with potential kidney involvement (eg systemic lupus erythematosus)
- patients on ACEI/ARB or diuretics
- bladder outflow obstruction until treated and the stability of the eGFR is established

All patients at risk of CKD should have a one-off quantitative proteinuria test (ACR or PCR). This does not need to be monitored unless there is clinically significant proteinuria (see **Q29**)

36. How often does eGFR need to be monitored in people with CKD?

1. Annually in all at risk groups (see **Q35**)
2. During intercurrent illness and peri-operatively in all patients with CKD.
3. Exact frequency should depend on the clinical situation. The frequency of testing may be reduced where eGFR levels remain very stable** but will need to be increased if there is rapid progression.

Stage	eGFR range (ml/min/1.73 m ²)	Typical testing frequency
1 and 2 (not part of the QOF)	≥ 60 + other evidence of kidney disease	12 monthly
3A and 3B	30–59	6 monthly
4	15–29	3 monthly**
5	<15	6 weekly

Table: 4: Measurement of eGFR: how often?

See:

www.nice.org.uk/Guidance/CG73/NiceGuidance/pdf/English

Or the Quick reference guide at:

guidance.nice.org.uk/CG73/QuickRefGuide/pdf/English

***Where Stage 4 disease is clearly non-progressive (in authors' experience often elderly people) in asymptomatic people with less than two years stable eGFR/creatinine, six month review is adequate.*

Referral in CKD

37. How should referral policy be developed locally?

We would expect that all primary care organisations bring together stakeholders representing patients, renal specialists, and primary care to develop a care pathway for the management of CKD.

38. Which patients should be referred for a renal opinion?

Again, please refer to the NICE CKD guideline documents available at: www.nice.org.uk/Guidance/CG73/NiceGuidance/pdf/English

Or the Quick reference guide at: guidance.nice.org.uk/CG73/QuickRefGuide/pdf/English

Referral is indicated in the following situations:

- Acute Kidney Injury (AKI); the discovery of an abnormal eGFR should prompt a review of historical eGFR and where eGFR is not available creatinine measurements
- All those with Stage 4 and 5 disease should have their care plan formally discussed with a specialist. It may be possible in some cases for assessment and follow up to take place at the GP practice. However guidance for monitoring and future referral or re-referral should be made explicit
- Higher levels of proteinuria (ACR ≥ 70 mg/mmol or PCR ≥ 100 mg/mmol) unless known to be due to diabetes and already appropriately treated
- Persistent invisible (microscopic) haematuria and proteinuria (ACR ≥ 30 mg/mmol or PCR ≥ 50 mg/mmol)
- Progressive CKD. NICE have defined progressive CKD by a fall in eGFR of ≥ 5 ml/min/1.73m² within one year (based on at least three readings) or a fall of ≥ 10 ml/min/1.73m² within five years
- Hypertension which remains poorly controlled despite the use of at least four antihypertensive drugs at therapeutic doses
- Renal anaemia (Hb < 11g/dl and thought to be due to CKD)
- Patients who present with a rare or genetic cause of renal disease (e.g. Adult Polycystic Kidney Disease – APKD)
- Suspected renal artery stenosis

In certain circumstances referral to another specialist would be appropriate initially. Elderly people with complex multiple problems may benefit from initial assessment by a geriatrician. Persistent invisible haematuria (with or without proteinuria) should also always prompt the urological exclusion of urinary tract malignancy in the over 50s. Once malignancy and other urological pathology are excluded, these patients can have their CKD managed by their GP in the usual way.

Where practices are experiencing difficulties accessing specialist renal advice then they should discuss this with the Commissioning Director.

Advice should be sought from the renal clinic regarding people with newly diagnosed Stage 4 CKD unless renal function is known to be stable. People who are frail and stable in Stage 4 or 5 and thought to be in the last year of life or who choose the no dialysis option should be recorded as needing support and palliative care without referral.

Some patients (approximately 15 to 20% or those approaching ESRD) choose conservative kidney care or the no dialysis option. This choice should be the results of careful shared decision making. This decision should be made with health care professionals familiar with all the dialysis modality options. Patients in these circumstances may still benefit from treatment of renal anaemia and need care and medicines management. On average people who choose conservative kidney care live about two years.

Further information on end of life care can be found at the NHS Evidence, Kidney care:

www.library.nhs.uk/kidney/SearchResults.aspx?tabID=288&catID=11918&

and

www.kidneycare.nhs.uk/i/assets/EoLC_Jun09.pdf

39. Who needs urgent referral?

Patients with newly diagnosed Stage 5 CKD should be referred urgently unless it is part of a known terminal illness or they are stable with a known management plan.

Urgent referral is required for acute renal failure (Acute Kidney Injury), malignant hypertension, hyperkalaemia ($K^+ > 7\text{mmol/L}$), severe uraemia, fluid overload, and nephrotic syndrome.

40. What information is required in a referral to a nephrologist?

- List of dates and results of previous serum creatinine measurements to assess stability, (many GP computer systems allow a list to be created of previous creatinine measures and then printed off or pasted into a referral letter).
- Serum potassium
- Haemoglobin
- Past medical and full drug history
- Blood pressure
- ACR or PCR values
- Urinalysis results
- Renal ultra sound if progressive disease, advanced (Stage 4 or 5) disease, visible or persistent invisible haematuria, those over 20 years of age with a

family history of polycystic kidney disease, or those with a palpable bladder/lower urinary tract symptoms.

- If diabetic: HbA1c results and evidence of other diabetic complications
- If prostate disease: details.

Practices should also refer to any local guidance as to the information required to support referral.

41. When is it reasonable NOT to refer someone to the renal unit?

Patients' wishes and co-morbidities should be taken into account when considering referral. It may not always be appropriate to refer people who are frail and stable in Stage 4 or 5 and thought to be in the last year of life. Though, these patients may still benefit from treatment of their renal anaemia.

42. How should I provide end of life care (EoLC) in CKD

GPs may be involved in end of life care (EoLC) in CKD. Most important is that time should be set aside for adequate discussion and exploring our patients wishes. People receiving EoLC in CKD should be placed on the cause for concern register or identified using other appropriate flags. Anaemia control may be important to reduce symptoms in EoLC in CKD.

Further information on EoLC can be found at:
www.kidneycare.nhs.uk/i/assets/EoLC_Jun09.pdf

QOF issues, computer reporting and exception reporting

43. Why is CKD included in the GP contract Quality and Outcomes Framework (QOF)?

There is evidence that the management of the CVD risk factors of people with CKD is not always optimal. People with CKD can readily be identified if pathology labs estimate GFR when they measure creatinine. There is strong evidence that good blood pressure control in patients with CKD alters their outcome. Hence blood pressure control became one of the QOF targets from 1st April 2006

The QOF has been remarkably successful in raising awareness of and improving the management of CKD.

44. When should I add someone to the CKD disease register?

The QOF CKD disease register is for people with Stage 3 to 5 CKD, i.e. the three more serious of the five stages of CKD, an eGFR $< 60\text{ml/min/1.73m}^2$.

Most GP computer systems do not automatically add people with a low eGFR to the disease register. This is because to diagnose CKD two readings of eGFR $< 60\text{ml/min/1.73m}^2$ at least three months apart are required (without an intermediate reading $\geq 60\text{ml/min/1.73m}^2$). If the criteria for diagnosing CKD are met but the two eGFR readings are in different stages of disease, then the class of CKD is determined by the higher (the least serious) reading.

Having determined the person has CKD you next need to go on and 'code' the diagnosis. Most of the UK currently uses "5-Byte" Read codes (Q45).

45. What code should I use for CKD?

The codes for CKD with and without proteinuria are set out in the 1Z... part of the Read code hierarchy. For which codes to use see the table below. For a more detailed discussion about the layout of the codes and where this code sits in the hierarchy please see the next question.

Please only use the 'with proteinuria' codes where a quantitative proteinuria test confirms proteinuria is present at clinically significant levels (ie ACR $\geq 30\text{mg/mmol}$ or PCR $\geq 50\text{mg/mmol}$ – see Q29). Do not base decisions about a diagnosis of proteinuria on urinary dipstick tests.

Please only use the codes in the table and migrate people coded as Stage 3 to either 3a or 3b.

Rubric	Code	Notes	Use
Codes which do not specify whether there is proteinuria – only use early in diagnosis before carrying out ACR test			
CKD stage 1	1Z10.	Note break in sequence as originally just one CKD3 code	Use a CKD code without specifying proteinuria at the stage you have at least two readings less than three months apart.
CKD stage 2	1Z11.		
CKD stage 3a	1Z15.		
CKD stage 3b	1Z16.		
CKD stage 4	1Z13.		
CKD stage 5	1Z14.		
Codes to use when there is no significant proteinuria ACR < 30 mg/mol (or PCR < 50 mg/mmol) in people without diabetes			
CKD stage 1 no proteinuria	1Z18.	Note sequence is alternate alphanumeric as the intermediary value is with proteinuria There are two sequence breaks one for just Stage 3 (do not use) and one for no I in the letter sequence	Use a CKD and no proteinuria code for people without clinically significant proteinuria (ACR test is < 30 mg/mmol or PCR < 50 mg/mmol)
CKD stage 2 no proteinuria	1Z1A.		
CKD stage 3a no proteinuria	1Z1E.		
CKD stage 3b no proteinuria	1Z1G.		
CKD stage 4 no proteinuria	1Z1J.		
CKD stage 5 no proteinuria	1Z1L.		
Codes to use where a person with CKD has proteinuria ACR ≥ 30 mg/mmol or PCR ≥ 50 mg/mmol			
CKD stage 1 with proteinuria	1Z17.	Note break in sequence as originally just one CKD3 code	Use a CKD and proteinuria code where there is a quantitative lab confirmation of clinically significant proteinuria. (ACR test is ≥ 30 mg/mmol or PCR > 50 mg/mmol)
CKD stage 2 with proteinuria	1Z19.		
CKD stage 3a with proteinuria	1Z1D.		
CKD stage 3b with proteinuria	1Z1F.		
CKD stage 4 with proteinuria	1Z1H.		
CKD stage 5 with proteinuria	1Z1K.		

Table 5: Table of recommended Read Codes (Significant proteinuria levels are for people without diabetes)

46. Why do people think that CKD has the wrong type of hierarchy in the wrong section of the Read code/terminology?

There has been a surprising amount of correspondence about this issue – and the points raised are good ones, and probably like many things were these codes to be reset out they would not be done in this way. The codes clinicians need are all there and the detail is set out in the question above.

However, for those who are interested in informatics and coding there are three basic problems:

- The codes do not sit where you would expect within the 'genitourinary disease' chapter. In this chapter K04 is the code for 'acute renal failure' and K05 is for 'chronic renal failure', with K050 for 'end stage renal failure.' Here or as part of K06 (renal failure unspecified) or its child code K060 (renal impairment) is the sort of place you expect to find CKD. Not in Chapter 1 the 'symptoms' chapter.
- The hierarchy would be better if the 'with proteinuria' or without proteinuria' were child codes of the same parent.
- Placing Stages 3a and 3b 'on the end' of one part of the hierarchy and in the middle of another can be confusing.

However, Read can be unclear in other areas. For example: the H3 (COPD) and H33 (Asthma) have a wholly inappropriate parent-child relationship¹¹; and the limitations in our ability to record data and investigations in osteoporosis¹² are even more problematic. Although not set out in the easiest order to follow, we can code everything we need to.

To discuss or debate the above outlined issues in further detail, please write to Simon de Lusignan at Informatics in Primary Care (EditorIPC@googlemail.com).

47. Who could/should be exception reported?

The usual categories of exception codes apply: 'patient unsuitable' and 'informed dissent'. There may be few situations where these apply other than in terminal illness where renal function may fail and there is no benefit to the patient in attempting to manage their renal impairment.

People who are intolerant to angiotensin converting enzyme inhibitors (ACE-I) and angiotensin II receptor blockers (ARB) are effectively exception reported as they are removed from the target population. The maximum tolerated dose of antihypertensive code has the same effect.

In the absence of proteinuria it is unnecessary to use ACE-I and ARB if blood pressure control is satisfactory.

The proportion of patients needed to be treated to achieve the target payment has been set so that there should be a very limited need to exception report.

¹¹ Faulconer ER, de Lusignan S. *An eight-step method for assessing diagnostic data quality in practice: chronic obstructive pulmonary disease as an exemplar*. Inform Prim Care. 2004;12(4):243-54.

¹² de Lusignan S, Chan T, Wood O, Hague N, Valentin T, Van Vlymen J. *Quality and variability of osteoporosis data in general practice computer records: implications for disease registers*. Public Health. 2005;119(9):771-80.

48. What should GPs record in their computer system?

Recording of all creatinine measures, cardiovascular risk factors (blood pressure, cholesterol and smoking habit) and evidence of renal damage, including the presence of proteinuria, are all important. Haemoglobin should be measured and recorded as some people with CKD become anaemic. A family history of CKD should also be recorded.

Negative results, particularly negative urine proteinuria tests and renal tract imaging results, will help with more sophisticated sorting of patients at a later date.

Diagnosis codes are not assigned automatically in many computer systems. So whilst eGFR results may be incorporated into records once pathology results are filed these patients may not be assigned a diagnostic code. You should not assign a diagnostic code based on a single eGFR reading as strictly at least two eGFR readings a minimum of three months apart are required for a diagnosis of CKD to be made. The class of CKD is based on the best (i.e. highest) eGFR reading during this period.

Sometimes when there is only one eGFR reading there are many more historic creatinine readings. If there are a series of similar serum creatinine values which are similar to the current result it may be reasonable to assign a class of CKD.

Finally, the stage of CKD should only be qualified as with or without proteinuria when proper quantitative measures (i.e. ACR or PCR) have been made and NICE threshold values exceeded (see **Q28**).

49. How do I get more information?

As a practice or locality consider talking to the nephrologist or unit that covers your geographical patch. Many produce local guidance and most are happy to come and talk/meet with local practice or locality groups. Also consider asking your local GP tutor to include management of CKD in your postgraduate programme and update courses.

Again, please refer to the NICE CKD guideline documents available at:
www.nice.org.uk/Guidance/CG73/NiceGuidance/pdf/English

Or the Quick reference guide at:
guidance.nice.org.uk/CG73/QuickRefGuide/pdf/English

Also, a comprehensive set of resources to help manage CKD, including links to calculators and guidelines is available at:
www.renal.org

50. Is any patient information available?

Patients may find the comprehensive information available from NICE helpful:
www.nice.org.uk/Guidance/CG73/

A patient information leaflet can be downloaded from:
www.renal.org/eGFR/resources/PatientCKDinf/an2007.pdf

Map of Medicine:
www.nhs.uk/pathways/kidneydisease/pages/avoiding.aspx

Management of CKD – summary

51. I need an overview of CKD management, it seems very complex?

Two factors largely determine the level of disease in CKD: the stage of CKD and the degree of proteinuria (measured quantitatively using ACR). Therefore as renal function declines so management needs to be more aggressive and referral is more likely. Likewise, as proteinuria increases so more active management is required.

Two tables seek to set out the principles of management of CKD. One is for people with CKD who do not have diabetes and one for people with CKD and diabetes. Areas where primary care management is appropriate (generally early stages of CKD with no or little proteinuria) are marked in green. Areas where primary or secondary care may be appropriate depending on the precise clinical scenario are coloured orange; and areas where specialist management is nearly always required in red.

	eGFR Best over 3/12 period	All CKD general management	ACR <30 mg/mmol	ACR 30 to 69 mg/mmol	ACR ≥ 30 mg/mmol + haematuria	ACR ≥70 mg/mmol
1	>90 + renal damage	All stages lifestyle advice: CVS risk FH Low salt & low K+ diet	No CVD no other CKD risk – no action	Use ACE / ARB Control BP >140/90		Manage BP with up to 4 agents – prior to referral -Send all SCr - Investigate US, Hb, bone
2	60-89 + renal damage		CKD risk – - repeat 1 yr	Ideally systolic 120-39		
3a	45-59	Obstruction in men	if BP <140/90 no need to change agents	CVD statin + usual 1 ^o prevention		
3b	30-44	Check Hb (<11g/dl)	Check 6/12			
4	15-29	Bone Ca PO4 Bone density	Manage in 1 ^o care if stable / refer if uncertain	Refer: To appropriate specialist -Cardiologist, Urologist, Diabetologist, Renal physician, Palliative care		
5	<15 or renal replacement			Appropriate pre-referral work-up		

Table 6: Managing CKD in a nutshell – for people with CKD but no diabetes

	eGFR Best over 3/12 period	All CKD DM patients	ACR ≤ 2.5 Men ≤ 3.5 Women	ACR > 2.5 Men > 3.5 Women
1	>90 + renal damage	All stages lifestyle advice: CVS risk FH Low salt – low K+ diet Obstruction in men	No CVD no other CKD risk – <i>no action</i>	Use ACE / ARB Control BP >130/80
2	60-89 + renal damage		CKD risk – – <i>repeat 1 yr</i>	Ideally systolic 120-29 Ideally diastolic <80
3a	45-59	Check Hb (<11g/dl) Bone Ca PO4 Bone density	Control BP	Use ACE / ARB even if no hypertension CVD statin + usual diabetes management
3b	30-44		Check 6/12	
4	15-29		Manage in 1 ^o care if stable / refer if uncertain	
5	<15 or renal replacement	Refer: To appropriate specialist -Cardiologist, Urologist, Diabetologist, Renal physician, Palliative care		

Table 7: Managing CKD in a nutshell – for people with CKD and diabetes

For further information please refer to the guidance:
www.nice.org.uk/Guidance/CG73/NiceGuidance/pdf/English

Or the Quick reference guide at:
guidance.nice.org.uk/CG73/QuickRefGuide/pdf/English

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