North of Tyne/Gateshead guidelines for detection, management and referral of adults with kidney disease

An electronic version of this document can also be viewed / downloaded from the North of Tyne and Gateshead Area Prescribing Committee Website at:

http://medicines.necsvu.nhs.uk/download/ckd-guidelines/

<table>
<thead>
<tr>
<th>Review date</th>
<th>Medicines Use and Guideline Group recommended review date: March 2019</th>
</tr>
</thead>
<tbody>
<tr>
<td>Membership of the guideline development group</td>
<td></td>
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</tbody>
</table>

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North of Tyne/Gateshead guidelines for detection, management and referral of adults with kidney disease

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Introduction
The previous North of Tyne guidelines were issued in April 2009 and were due for review in January 2012. Several national guidelines have been updated since that time, both on chronic kidney disease (CKD) and on acute kidney injury (AKI). The current revision aims to ensure that these guidelines are implemented within the local context and to include Gateshead.

The guideline applies to adults (aged ≥18y). It is a guideline, and as with all guidelines, its use should be interpreted in the clinical context: no guideline can cover all clinical situations. It is written for healthcare professionals and is designed to support shared decision-making, but is not a substitute for the provision of personalised information about the risks and benefits of a particular decision, and cannot take fully into account the values and preferences of individual patients.

Although most CKD is associated with age- and hypertension-related kidney damage, and most AKI reflects acute intercurrent illness, it remains vital that glomerulonephritis, interstitial nephritis, and renal involvement in systemic vasculitis are recognised and referred without delay. If in doubt, GPs should seek advice from the Renal Unit by fax or letter. Letters should contain all relevant clinical information, including current and past drug treatment, all previous measurements of serum creatinine and eGFR (using the cumulative results function in ICE – NB nephrologists can only access one result at a time on ICE OpenNet), and the question to be answered. The renal SpR on call should only be telephoned for advice when emergency admission directly to the renal ward is being considered.

The guideline draws on NICE guidelines and other national guidelines.

Disclaimer
In making recommendations for drug treatment, it is assumed that clinicians will exclude contraindications and interactions, referring to the BNF and local formularies as necessary, when managing individual patients.
Abbreviations used in this document

ACEI: Angiotensin Converting Enzyme Inhibitor
ACR: Albumin:Creatinine Ratio
ADHF: Acute Decompensated Heart Failure
ADPKD: Autosomal Dominant Polycystic Kidney Disease
AKI: Acute Kidney Injury
ARB: Angiotensin II Receptor Blocker
BKPA: British Kidney Patient Association
BMD: Bone Mineral Density
BP: Blood Pressure
CCB: Calcium Channel Blocker
CKD: Chronic Kidney Disease
CKD-EPI: Chronic Kidney Disease – EPIdemiology collaboration
CVD – cardiovascular disease
DEXA: Dual Energy Xray Absorptiometry
eGFR: estimated Glomerular Filtration Rate (in ml/min/1.73m²)
ESC: European Society of Cardiology
FRAX: Fracture Risk Assessment Tool
Hb: Haemoglobin
JVP: Jugular Venous Pressure
MDRD: Modification of Diet in Renal Disease
MRA: Mineralocorticoid Receptor Antagonist
NHSE: NHS England
NICE: National Institute of Health and Care Excellence
NSAID: Non-Steroidal Anti-Inflammatory Drug
PCR: Protein:Creatinine ratio
POCT: Point of Care Testing
SIADH: Syndrome of Inappropriate AntiDiuretic Hormone secretion
U&E: Urea, Creatinine and Electrolytes
UTI: Urinary Tract Infection
Summary of reasons to consider referral to Secondary care

For immediate admission (usually via local general medical take)
1. Suspected systemic vasculitis with renal involvement
2. Acute Kidney Injury stage 3
3. Severe hyperkalaemia (serum K > 6.5 mmol/L)
4. Accelerated hypertension (BP > 180/110 mmHg with signs of papilloedema or retinal haemorrhage). (NB patients with suspected phaeochromocytoma (labile or postural hypotension, headache, palpitations, pallor and sweating) should ideally be admitted to the local Endocrine unit)

For urgent outpatient review
5. Nephrotic syndrome – oedema, hypoalbuminaemia, and heavy proteinuria
6. Recent Acute Kidney Injury stage 1-2 not attributable to sepsis, hypovolaemia or hypotension

For routine Nephrology outpatient review
7. Chronic kidney disease stage G4 or above (i.e. eGFR < 30)
8. Sustained decrease in eGFR of ≥ 15, or of ≥ 25% if initial eGFR ≤ 60, within 12 months
9. ACR >= 70 mg/mmol, unless known to be caused by diabetes and already appropriately treated (e.g. already on the maximal tolerated dose of an ACEI or ARB)
10. ≥ 30% rise in serum creatinine (or ≥ 25% fall in eGFR) on initiation or up-titration of ACEI or ARB after exclusion of alternative causes e.g. hypovolaemia
11. Asymptomatic non-visible haematuria combined with urine albumin:creatinine ratio > 30 mg/mmol, irrespective of eGFR

Anaemia (Hb < 105 g/L) in the presence of CKD stage G3a-G5
after exclusion of other causes and after a trial of oral iron unless serum ferritin is already > 100 micg/L and transferrin saturation > 20%; or if oral iron not tolerated.

For review in the renal genetics clinic
12. Suspected metabolic stone disease (see flow chart)
13. Suspected or proven polycystic kidney disease
14. Suspected inherited renal disorder, including renal tubular acidosis, congenital abnormality of the kidney and urinary tract, familial haematuria

Consider referral for specialist review
15. Resistant hypertension (BP > 140/90 mmHg despite maximal tolerated doses of a 3-drug combination according to the NICE AB/CD algorithm)
16. Early onset hypertension without obvious cause (age < 40y)

These are guidelines, not rules. The value added from referral may relate to current management, or to advanced decision-making, particularly amongst those at risk of progression to CKD stage G5. Please phone the Renal Specialist Registrar on call only to seek urgent admission. If admission is required, depending on the clinical situation and the bed state, you may be asked to send the patient to your local Ambulatory Care facility, Emergency Department, or the Emergency Admissions Suite at the Freeman Hospital. For all other questions, send a request for ‘Advice and Guidance’ or a referral letter.
Summary of monitoring and management of CKD and AKI

Diagnosis and classification of Chronic Kidney Disease
Use the NICE CKD classification to classify CKD (see table 1).

Measurement of eGFR and of urine albumin:creatinine ratio
eGFR should be measured at least annually in

- patients taking ciclosporin
- patients taking tacrolimus
- patients taking lithium
- patients taking regular non-steroidal anti-inflammatory drugs

eGFR and ACR should be measured at least annually in

- patients with chronic kidney disease
- patients with diabetes
- patients with hypertension
- patients with cardiovascular disease
- patients with a history of acute kidney injury within the past 3 years
- patients with multisystem disease with potential renal involvement, e.g. SLE, vasculitis
- patients with structural renal tract disease, recurrent kidney stones, or prostatic hypertrophy
- patients with family history of premature CKD G5 or hereditary kidney disease
- patients in whom there has been opportunistic detection of haematuria.

eGFR should be measured at the intervals given in Figure 2 for monitoring of patients with chronic kidney disease. ACR should be measured at diagnosis and annually thereafter in all patients with CKD: whether to measure ACR more frequently than annually should be decided on a case-by-case basis, with additional measurements only if the results are likely to change management – e.g. if blood-pressure-lowering treatment is being titrated in order to reduce albuminuria.

Measurement of serum creatinine
Ask patients to avoid eating meat and taking intense muscular exercise for 12h prior to the serum creatinine blood test.

Interpret measurements of serum creatinine with care if the patient has been taking Trimethoprim or Cimetidine in the 14 days prior to the test: these drugs can cause significant increases in serum creatinine from baseline, particularly in patients with pre-existing CKD, without any change in true GFR.

Measurement of urine albumin:creatinine ratio
To detect proteinuria, request urine Albumin:Creatinine ratio (ACR). Urine Protein:Creatinine ratio (PCR) can be used as an alternative for monitoring of known CKD if urine ACR > 70 mg/mmol. If urine ACR is between 3 mg/mmol and 70 mg/mmol, this should be confirmed on an early morning urine sample. If ACR > 70 mg/mmol, a repeat sample need not be tested. NB urine infection does not need to be excluded before measurement of ACR: bacteriuria can increase total urine protein but does not increase urine albumin unless there is systemic disease.
Detection of haematuria
Use dipstick tests to detect non-visible haematuria. Evaluate further if there is a result of 1+ or more. Do not use microscopy to confirm a positive result.

Renal ultrasound scanning
Request an ultrasound scan of the kidneys and urinary tract in patients who have rapid deterioration of kidney function (sustained fall of eGFR > 15 or of ≥ 25% if initial eGFR ≤ 60, over 12 months or less); visible or persistent non-visible haematuria; symptoms of urinary tract obstruction; a family history of polycystic kidney disease and are aged over 20 years; have an eGFR < 30; are considered by a nephrologist to require a renal biopsy.

Measurement of haemoglobin
Measure Hb at least annually to look for renal anaemia in patients with CKD G3b, G4 and G5.

Measurement of serum calcium, phosphate, and albumin
Do not routinely measure calcium, phosphate and albumin in patients with CKD G3a or in stage G2 or G1 (i.e. in patients with eGFR > 45). Measure calcium, phosphate, and albumin alongside measurements of eGFR when monitoring patients with CKD3b or higher (i.e. in patients with eGFR < 45).
**Definition and classification of CKD**

Chronic kidney disease should be diagnosed and classified according to the NICE criteria, which are based on estimated Glomerular Filtration Rate, (eGFR) and on markers of kidney damage. To qualify as CKD, a patient should have evidence of persistent albuminuria; glomerular haematuria; evidence of structural abnormalities on renal imaging; evidence of renal abnormalities on renal biopsy; a kidney transplant; or eGFR < 60 for at least 3 months, without any intervening measurements >60.

Patients diagnosed with CKD should have this classified using the criteria in the table below, with the information included in the clinical notes, and reviewed annually (or more frequently if appropriate).

<table>
<thead>
<tr>
<th>GFR categories (mL/min/1.73 m²), description and range</th>
<th>ACR categories (mg/mmol), description and range</th>
<th>A1</th>
<th>A2</th>
<th>A3</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥90 Normal and high</td>
<td>G1</td>
<td>No CKD in the absence of markers of kidney damage</td>
<td></td>
<td></td>
</tr>
<tr>
<td>60–89 Mild reduction related to normal range for a young adult</td>
<td>G2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>45–59 Mild–moderate reduction</td>
<td>G3a'</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30–44 Moderate–severe reduction</td>
<td>G3b</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15–29 Severe reduction</td>
<td>G4</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;15 Kidney failure</td>
<td>G5</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

Table 1. Classification of CKD, taken from NICE CG182.
### Frequency of monitoring of CKD, according to CKD stage

<table>
<thead>
<tr>
<th>GFR categories (ml/min/1.73 m²), description and range</th>
<th>A1 &lt;3 Normal to mildly increased</th>
<th>A2 3–30 Moderately increased</th>
<th>A3 &gt;30 Severely increased</th>
</tr>
</thead>
<tbody>
<tr>
<td>G1 ≥90 Normal and high</td>
<td>≤1</td>
<td>1</td>
<td>≥1</td>
</tr>
<tr>
<td>G2 60–89 Mild reduction related to normal range for a young adult</td>
<td>≤1</td>
<td>1</td>
<td>≥1</td>
</tr>
<tr>
<td>G3a 45–59 Mild–moderate reduction</td>
<td>1</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>G3b 30–44 Moderate–severe reduction</td>
<td>≤2</td>
<td>2</td>
<td>≥2</td>
</tr>
<tr>
<td>G4 15–29 Severe reduction</td>
<td>2</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>G5 &lt;15 Kidney failure</td>
<td>4</td>
<td>≥4</td>
<td>≥4</td>
</tr>
</tbody>
</table>

Table 2. Frequency of monitoring of CKD according to CKD stage, taken from NICE CG182. The figures in the boxes refer to the number of times per year that tests should be repeated. However, this frequency should be tailored to the individual according to the underlying cause of CKD, the past pattern of progression, co-morbidities (especially heart failure), changes to drug treatment (especially ACEI, ARB and diuretics), intercurrent illness, and whether the patient has chosen conservative management.
Management of patients with chronic kidney disease

Provide patients diagnosed with CKD with individualised advice and information on the diagnosis

- risk of cardiovascular disease
- risk of progressive kidney disease, often asymptomatic until advanced
- mortality of established renal failure = similar to many cancers
- increased risk of acute kidney injury during intercurrent illness

Offer patients the Kidney Care UK factsheet on CKD or Kidney Research UK’s information booklet

Offer patients with CKD the Kidney Care UK/NHS England/RCGP advice sheet on patients at risk of AKI ‘How to keep your kidneys safe’

Provide patients diagnosed with CKD with individualised advice on what to do during acute illness, including

- drugs to stop (e.g. antihypertensive drugs if hypotensive: diuretic drugs if hypovolaemic; drugs that are renally cleared if risk of AKI (including sulphonylureas, Metformin); NSAIDs
- need for early clinical assessment and repeat blood tests

Do not use a risk assessment tool to assess cardiovascular disease (CVD) risk in patients with an eGFR < 60 or with an ACR > 30 mg/mmol. Offer Atorvastatin 20mg for primary prevention of CVD. Increase the dose if necessary to achieve ≥40% reduction in non-HDL cholesterol. Discuss with specialist before increasing dose if eGFR < 30 (see FATS guidelines).

Offer treatment for cardiovascular risk factors, taking into account the patient’s views and preferences. If the patient wishes to minimise risk of future cardiovascular disease and progression of kidney damage,

- advise on lifestyle
  - weight loss if obese
  - regular aerobic exercise (even if it does not result in weight loss)
  - reduce dietary salt
  - increase dietary potassium (fruit and vegetables) unless eGFR < 30 or K > 4.5 mmol/L
  - smoking cessation advice
- offer BP-lowering treatment (for target BP, see section on BP management)
- offer anti-platelet agents to patients with established cardiovascular disease according to local guidelines
Anaemia management

Measure Hb at least annually to allow detection of renal anaemia in patients with CKD G3b, G4 and G5.

Do further investigations if the Hb < 110 g/L or the patient has symptoms of anaemia and Hb < 130 g/L. Consider and rule out haemolysis, iron deficiency, B12 and folate deficiency, and active bleeding.

If the patient has a normochromic normocytic anaemia with Hb < 110 g/L and eGFR < 60, check iron status using serum ferritin and transferrin saturation.

Offer oral iron supplementation to maintain serum ferritin > 100 micg/L and transferrin saturation >20% (unless serum ferritin > 800 micg/L).

Refer to renal services if
eGFR < 60 and Hb < 110 g/L AND

- ferritin > 100 micg/L and transferrin saturation > 20%
- patient does not tolerate oral iron or ferritin is <100 micg/L or transferrin saturation <20% despite a 3-month trial of oral iron

Once the patient has been evaluated in the Nephrology clinic, the nurse-led Renal Anaemia Team will co-ordinate treatment with intravenous iron and subcutaneous darbepoetin (darbepoetin is the preferred erythropoiesis stimulating agent locally), adjusted according to measurements of Hb and iron status performed in primary care.
Blood pressure management

Drug treatment should always be on the background of repeated lifestyle advice on cardiovascular risk (smoking cessation, exercise, weight reduction if obese, reduction in dietary salt intake).

Offer all patients the chance to discuss the risk, treatment burden, and potential benefit of BP reduction using a risk estimator, e.g. Qintervention.

For patients with co-morbidities likely to alter the risk-benefit ratio of BP reduction (e.g. orthostatic hypotension from autonomic neuropathy; falls attributed to hypotension), decide an individualised BP target range.

Unless there is a good clinical or patient-choice-related reason otherwise, use these targets for BP:

<table>
<thead>
<tr>
<th>Patients aged &lt; 80y</th>
<th>Clinic-measured BP</th>
<th>Home or ambulatory BP</th>
</tr>
</thead>
<tbody>
<tr>
<td>CKD, no diabetes</td>
<td>SBP &lt; 140, DBP &lt; 90</td>
<td>SBP &lt; 135, DBP &lt; 85</td>
</tr>
<tr>
<td>CKD, with diabetes and patients with urine ACR &gt; 70 mg/mmol</td>
<td>SBP &lt; 130, DBP &lt; 80</td>
<td>SBP &lt; 125, DBP &lt; 75</td>
</tr>
</tbody>
</table>

For patients over 80, allow a 10 mmHg higher SBP:

<table>
<thead>
<tr>
<th>Patients aged &gt;80</th>
<th>Clinic-measured BP</th>
<th>Home or ambulatory BP</th>
</tr>
</thead>
<tbody>
<tr>
<td>CKD, no diabetes</td>
<td>SBP &lt; 150, DBP &lt; 90</td>
<td>SBP &lt; 145, DBP &lt; 85</td>
</tr>
<tr>
<td>CKD, with diabetes and patients with urine ACR &gt; 70mg/mmol</td>
<td>SBP &lt; 140, DBP &lt; 80</td>
<td>SBP &lt; 135, DBP &lt; 75</td>
</tr>
</tbody>
</table>

When treating hypertension, avoid reducing clinic BP to < 120 mmHg, and home BP to < 115 mmHg. If the patient has heart failure with reduced ejection fraction, seek specialist advice from a nephrologist or heart failure clinic before reducing doses of drug treatments that improve prognosis (ACEI, ARB, MRA) because of low SBP.

Choice of antihypertensive drug treatment

Follow the NICE algorithm for antihypertensive drug treatment unless the patient has a specific indication for an ACEI/ARB, namely:

- heart failure/LV systolic dysfunction
- diabetes mellitus with an ACR > 3 mg/mmol
- hypertension with an ACR > 30 mg/mmol
- ACR of > 70 mg/mmol irrespective of blood pressure
Changes in kidney function during ACEI/ARB/diuretic treatment in primary care

This advice applies to monitoring of pharmacotherapy in clinically stable patients; it does NOT apply to patients with intercurrent acute illness.

Measure urea, creatinine, and electrolytes (U&Es) before initiation or up-titration and again within 2 weeks of initiation or up-titration of dose of ACEI, ARB, or diuretic. Use the immediate pre-treatment serum creatinine concentration as the baseline. In patients with heart failure, measure U&Es within 1 week of initiation or up-titration of spironolactone or eplerenone (Mineralocorticoid Receptor Antagonists, MRAs), then monthly for the first 3 months, and 3-monthly for 1 year, and 4-monthly thereafter.

Kidney function: ACEI and ARB

If serum creatinine rises by >15% but <30% from initial baseline

- continue but repeat U&Es in a further 1 to 2 weeks
- arrange clinical review including assessment of fluid status and blood pressure
  - try to continue ACEI/ARB treatment if there is a strong indication, e.g. heart failure, albuminuric CKD, history of myocardial infarction
  - reduce or stop other BP-lowering drugs (CCB, alpha blockers) if SBP < 120 mmHg
  - reduce concurrent diuretics if there is clinical evidence of hypovolaemia/overdiuresis

If serum creatinine increases at any point by ≥30% from baseline

- arrange clinical review including assessment of fluid status and blood pressure
  - try to continue ACEI/ARB treatment if there is a strong indication, e.g. heart failure, albuminuric CKD, history of myocardial infarction
  - reduce or stop other BP-lowering drugs (CCB, alpha blockers) if SBP < 120 mmHg
  - reduce concurrent diuretics if there is clinical evidence of hypovolaemia/overdiuresis
- if renal function does not return to baseline with these measures,
  - stop the ACEI or ARB, or
  - reduce the dose to a previously tolerated dose and recheck renal function in 5-7 days; add an alternative antihypertensive medication if required
- consider obtaining advice from nephrology, even if serum creatinine returns to baseline
- obtain advice from local heart failure specialist team if the indication for treatment was heart failure: continuing an ACEI/ARB in heart failure with reduced ejection fraction may be beneficial even if serum creatinine rises by >30%

Kidney function: Diuretics including MRAs (e.g. Spironolactone)

Increases in serum creatinine and urea are an expected consequence of haemoconcentration caused by diuretic treatment, and do not necessarily mean that the drugs have caused kidney damage. Treat the patient, not the blood test: repeated clinical examination is key, paying attention to avoidance of hypovolaemia and hypotension. Disproportionate rises in blood urea may reflect effective hypovolaemia and should prompt clinical reassessment.

Stop blood-pressure-lowering drugs that are not specifically indicated, or contraindicated, in heart failure (e.g. CCB, alpha blockers) if SBP < 120 mm Hg. Seek specialist advice from a heart failure team or nephrologist if concerned.
Potassium management

Hyperkalaemia is common in patients with CKD, particularly if they are receiving treatment with ACEI, ARB, MRA (e.g. spironolactone), or NSAIDs. Hyperkalaemia can cause cardiac arrest, often without warning symptoms, or muscle paralysis. Management in primary care depends on the severity of hyperkalaemia and on the clinical context. Hyperkalaemia is classified as follows:

- Severe hyperkalaemia = serum K $\geq$ 6.5 mmol/L
- Moderate hyperkalaemia = serum K 6.0-6.4 mmol/L
- Mild hyperkalaemia = serum K 5.5 – 5.9 mmol/L

Measurement of serum potassium
Serum K should be measured in patients with CKD (frequency depends on CKD stage, see table), in patients with heart failure, and within 1-2 weeks of initiation or an increase in dose of an ACEI, ARB, or MRAs. NB Hyperkalaemia may be artifactual in samples sent from primary care: this can be caused by fist clenching during phlebotomy, use of small-gauge needles causing low-grade haemolysis, prolonged tourniquet use, and, most importantly, delays in sample processing, particularly in cold weather.

Severe hyperkalaemia (K $\geq$ 6.5 mmol/L): refer to hospital (usually via A&E) for immediate assessment and treatment

Moderate hyperkalaemia (K 6.0-6.4 mmol/L): management depends on clinical context:

- If the patient is acutely unwell, or has AKI, stop the ACEI, ARB or MRA and refer to hospital for immediate assessment and treatment.
- If the patient is clinically stable (i.e. the test was done as a routine check rather than for acute illness, and there is no AKI warning stage test result), undertake medication review within 1 working day of the result. If hyperkalaemia is unexpected, consider arranging a repeat test the following day taking steps to minimise any of the factors that can cause artifactual hyperkalaemia.
  - Look for and remove other contributors to hyperkalaemia, including
    - Trimethoprim/CoTrimoxazole
    - Potassium supplements
    - Potassium-sparing diuretics (beware combinations with furosemide)
    - Use of salt substitutes e.g. ‘LoSalt’
    - NSAIDs
    - Non-selective beta-blockers
    - Digoxin toxicity
  - Review the patient clinically: reduce/stop diuretics if evidence of over-diuresis.
  - If the patient is on ACEI, ARB or MRA, stop immediately, repeat serum K within 1 week, and review indications
    - If used for hypertension, consider an alternative antihypertensive drug.
    - If used for heart failure with reduced ejection fraction or diabetic kidney disease with albuminuria, re-start at a lower dose once serum K < 5.5 mmol/L and then continue to monitor; if the patient was on a combination of ACEI/ARB and an MRA, only re-start one of these drugs at a time
o Provide patients with a diet advice sheet on reduction of potassium intake.
o If problems with hyperkalaemia persist, refer to renal medicine for dietetic advice
o Seek advice from local heart failure specialist team if the indication for treatment was heart failure

**Mild hyperkalaemia** (K 5.5-5.9 mmol/L): management depends on clinical context

- If the patient is acutely unwell, or has AKI, stop the ACEI, ARB or MRA and consider referral to hospital for immediate assessment and treatment.
- If the patient is clinically stable (i.e. the test was done as a routine check rather than for acute illness, and there is no AKI warning stage test result), undertake medication review within 1 working day of the result. If hyperkalaemia is unexpected, consider arranging a repeat test within 3 days, taking steps to minimise any of the factors that can cause artifactual hyperkalaemia
  o Look for and remove other contributors to hyperkalaemia, including
    ▪ Trimethoprim/CoTrimoxazole
    ▪ Potassium supplements
    ▪ Potassium-sparing diuretics (beware combinations with furosemide)
    ▪ Use of salt substitutes e.g. ‘LoSalt’
    ▪ NSAIDs
    ▪ Non-selective beta-blockers
    ▪ Digoxin toxicity
  o Review the patient clinically: reduce/stop diuretics if evidence of over-diuresis.
  o If the patient is on ACEI or ARB and/or MRA, halve dose of one or both, and review indications:
    ▪ If used for hypertension, consider an alternative antihypertensive drug.
    ▪ If used for heart failure with reduced ejection fraction or diabetic kidney disease with albuminuria, continue, but monitor carefully.
  o Provide patients with a diet advice sheet on reduction of potassium intake.
  o If problems with hyperkalaemia persist, refer to renal medicine for dietetic advice
  o Consider seeking advice from local heart failure specialist team if the indication for treatment was heart failure

This advice is based on the Renal Association/Resuscitation Council guideline on hyperkalaemia section on primary care (p78), on Think Kidneys Acute Kidney Injury guidance, on ESC guidelines, on British National Formulary, and on NICE Clinical Knowledge Summaries
Bone disease management in CKD

Measurement of calcium, phosphate, 25-hydroxyvitamin D and parathyroid hormone

Measure calcium, phosphate and albumin at least twice a year in patients with CKD stage G3b.

Measure calcium, phosphate, and albumin at least twice a year in patients with CKD G4

(For patients with stable stage G4 CKD discharged to primary care for monitoring, the nephrologist responsible for the discharge will specify if there is a requirement to monitor parathyroid hormone, and will specify a level at which to refer the patient back for management of hyperparathyroidism.)

Measure 25-hydroxyvitamin D

- If the serum calcium is below the lower limit of normal
- If the patient has had a fragility (low impact) fracture
- If osteomalacia or osteoporosis is suspected on clinical grounds
- If the patient is found to have reduced bone density on Bone Mineral Density scanning
- If treatment with an antiresorptive agent (bisphosphonate, denosumab) is planned
- Concurrently with measurements of calcium, phosphate, and albumin in patients with CKD

Alfacalcidol or calcitriol should usually only be initiated by, or in consultation with, a nephrologist or endocrinologist. Do not use measurements of 25-hydroxyvitamin D to adjust the dose of alfacalcidol or calcitriol. (These drugs provide fully activated vitamin D, which is present at far lower concentrations than the 25-hydroxyvitamin D substrate; regular measurements of serum calcium, phosphate and parathyroid hormone are required for dose adjustment).

Treat vitamin D deficiency and insufficiency according to local guidelines, irrespective of the stage of CKD.

Seek advice from a nephrologist if serum calcium or phosphate are abnormal.

Use FRAX or Qfracture tools to assess fracture risk.

If treatment for osteoporosis is considered necessary (e.g. after fragility fracture; low BMD on DEXA):

- follow local guidelines and dosage instructions in the BNF for patients with CKD G1-G3b
- note that oral bisphosphonates are all contraindicated in CKD G4 and G5; the only licensed treatment option is Denosumab
- avoid bisphosphonates in women planning a future pregnancy
- refer patients with CKD and osteoporosis to a consultant in metabolic bone disease
Detection of haematuria

Blood in the urine may be caused by inherited or acquired kidney disease, systemic disease affecting the kidney, and by a range of urological conditions including benign and malignant renal tumours, urothelial tumours, benign and malignant prostate disease, and urinary tract infections. The purpose of this guideline is to ensure that those patients who would benefit from specialist investigation for renal causes are appropriately investigated and referred.

Many patients with asymptomatic, non-visible haematuria are likely to have kidney disease – either a genetic disorder of type IV collagen (causing Alport’s syndrome or thin basement membrane nephropathy) or a mild glomerulonephritis (e.g. IgA disease). These diseases can only currently be diagnosed by kidney biopsy, which carries some risks. There are no specific treatments for either of these diseases. So at present it is not justified to offer renal biopsy unless there is evidence that the patient is at high risk of progressive kidney damage, or needs the biopsy for insurance or employment reasons.

NICE recommends that patients with asymptomatic, non-visible haematuria should not be referred to Nephrology unless they have a urine albumin:creatinine ratio > 30 mg/mmol or meet the eGFR-based referral criteria. However, patients with a family history of non-visible haematuria, particularly if there are family members with a history of chronic kidney disease, should be offered referral to the Renal Genetics Clinic.

The flow charts on the following page summarise the appropriate investigation of patients with visible and non-visible haematuria. This guidance is consistent both with NICE CKD guidance and with updated NICE Cancer Referral guidance.
Assessment and referral of patients with non-visible haematuria

Non-visible haematuria not due to symptomatic UTI

Age ≤ 45 years

Measure clinic blood pressure.
Send blood for creatinine, eGFR, electrolytes
Send urine for ACR
Ultrasound renal tract/bladder (ensure full bladder)

Investigations normal
BP < 140/90 AND
eGFR ≥ 60 ml/min/1.73m2 AND
ACR < 30 mg/mmol

Cause not established
Annual reassessment in primary care whilst haematuria persists:
Blood pressure, blood for creatinine, eGFR, electrolytes, urine dip for blood, urine for ACR
Refer to urology if:
Develops visible haematuria, or
Symptomatic non-visible haematuria
Refer to nephrology if:
Develops proteinuria
eGFR < 30 ml/min/1.73m2 on 2 separate occasions, or eGFR falls by ≥ 5
ml/min/1.73m2 over 1 year, or ≥ 10
ml/min/1.73m2 over 5 years

Investigations abnormal
BP > 140/90 OR
eGFR < 60 ml/min/1.73m2 OR
ACR ≥ 30 mg/mmol OR
Abnormality of renal tract on ultrasound

Cause not established
Refer to Nephrology if:
BP > 140/90 OR
eGFR < 60 ml/min/1.73m2 OR
ACR ≥ 30 mg/mmol
All others - urgent referral

Cause established and managed

Refer to Nephrology or Urology if abnormality of renal tract on ultrasound scan or if not optimal visualisation

Notes
Non-visible haematuria is confirmed with ≥ 1+ blood or more on urine dipstick, on 2 or more occasions as soon as possible and within 6 weeks (urine microscopy should not be used to diagnose haematuria).
Non-visible haematuria should not be attributed to oral anticoagulants in the therapeutic range and/or anti-platelet agents as a cause.
If associated with a UTI, treat UTI and ensure haematuria has resolved

Assessment and referral of patients with visible haematuria

Aged < 45 years
Visible haematuria without a symptomatic UTI, or visible haematuria recurs after successful treatment of UTI

Refer to urology
Urgent referral

Aged ≥ 45 years
Visible haematuria without a symptomatic UTI, or visible haematuria recurs after successful treatment of symptomatic UTI

Refer to urology
2 week pathway for suspected cancer

Notes
Visible haematuria should not be attributed to oral anticoagulants in the therapeutic range and/or anti-platelet agents as a cause.
Acute Kidney Injury

Acute kidney injury (AKI) is a clinical syndrome characterised by a sudden reduction in renal excretory function. The term does not imply physical injury to the kidneys. AKI is a strong marker of illness and potentially poor prognosis. Preventable AKI costs the NHS more than venous thromboembolism. The national definitions of AKI are based on changes in serum creatinine concentration over time and on urine output:

1. AKI stage 1 is a rise of > 1.5x the baseline level, or a rise of >26 micmol/L within 48h, or a urine output of <0.5 ml/kg/h for 6-12h
2. AKI stage 2 is a rise of > 2x baseline or a urine output <0.5 ml/kg/h for ≥ 12h
3. AKI stage 3 is a rise of > 3x baseline or a rise of > 1.5x baseline to > 354 micmol/L, a urine output of < 0.3 ml/kg/h for ≥ 24h, or anuria for ≥ 12h.

Biochemistry laboratories should issue one of the above ‘warning stage test results’ alongside measurements of serum creatinine concentration for patients whose results fulfil the criteria for AKI compared to their baseline serum creatinine. Extensive guidance for GPs on how to respond to these alerts is available on the ‘Think Kidneys’ website.

AKI warning stage test results must, like every other lab test, be interpreted in clinical context, and in particular with the knowledge of why the blood test was sent. They are best seen as a possible indicator that the patient is acutely unwell. The test result is NOT a diagnosis. False positive test results can result from

- misleadingly high current creatinine results
  - recent trimethoprim or cimetidine treatment
  - meat intake, strenuous muscular exertion
  - marked change in muscle mass since the baseline value
- misleadingly low baseline creatinine results
  - results taken during iv fluid therapy/volume overload
  - results taken during pregnancy
- progression of CKD- particularly if the ‘baseline’ creatinine was taken nearly a year before the current sample.

False positive and false negative test results can result from reliance on ‘point of care tests’ (POCT) to measure serum creatinine, and these test results will not be included in the database used by laboratories to generate warning stage test results: if AKI is suspected, a sample should be sent to a laboratory for definitive measurement.
Prevention of AKI

Detailed advice on identification of patients at risk is available on the Think Kidneys website (see p9 for detailed advice on prescribing). Avoidance of truly nephrotoxic drugs (e.g. gentamicin, amphotericin, cisplatinum) is largely a secondary care issue. During acute illness in patients at risk, temporary suspension of drugs that can impair renal perfusion (NSAIDs, diuretics, antihypertensive drugs including ACEI and ARB), and drugs that may accumulate due to reduced renal clearance (including Metformin, Digoxin, Lithium, and some opiates) is advised. Advice to patients to suspend their own treatment during acute fever and diarrhoea ('sick day guidance') should be tailored to the individual patient.

Management of patients who have had AKI

Detailed advice on management of AKI in primary care is available on the Think Kidneys website. This includes advice on medicines management of patients with, or recovering from AKI, including whether/when to restart antihypertensive drugs.

Offer all patients the BKPA/NHS England leaflet for patients who have had AKI.

Follow-up after an episode of AKI

- Follow advice in discharge summaries on follow-up measurements of kidney function.

- If no specific advice is given, ensure regular measurements of kidney function until it is clear whether or not the patient has made a full recovery (i.e. back to pre-admission eGFR).

- If recovery is incomplete, and the patient is left with Chronic Kidney Disease, manage accordingly.

- Even if recovery appears complete, a further test of eGFR and urine ACR should be undertaken at 3 months after discharge as a minimum.

- Continue follow-up with annual eGFR and urine ACR for a minimum of 2 years.
Laboratory measurements relating to kidney disease

Measurement of serum creatinine
As recommended in NICE CKD (CG182), laboratories should

- Use a creatinine assay that is zero-biased with respect to Isotope Dilution Mass Spectrometry, e.g. an enzymatic assay.
- Use the CKD-EPI equation to calculate eGFR\textsubscript{creatinine}
- Report the eGFR alongside all measurements of serum creatinine, irrespective of setting
  - Include a warning for results for inpatients that eGFR might not accurately reflect true GFR (because changes in serum creatinine concentration lag behind changes in GFR)
- Apply a correction factor to eGFR measurements using the CKD-EPI equation for people of African or African-Caribbean origin (multiply eGFR by 1.159), or if ethnic origin is not provided on the request, include this information on the report
- Participate in the UK National External Quality Assessment Scheme for creatinine
- Report eGFR to the nearest whole number

Measurement of urine albumin:creatinine ratio and protein:creatinine ratio
Laboratories should:

- Use ACR in preference to PCR for quantitation of proteinuria, unless the patient is already known to have ACR > 70 mg/mmol
- Recommend a repeat test on an early morning urine sample to confirm an initial measurement of ACR between 3 and 70 mg/mmol
- Use PCR in place of ACR if the urine albumin concentration in the sample is above the limit of detection despite 10-fold dilution

Measurement of Cystatin C
The guideline group debated at length whether to recommend Cystatin-C-based eGFR, which NICE recommended on the grounds that its use might reduce the false-positive diagnosis of CKD stage G3a, particularly amongst elderly women. To our knowledge, no other laboratory in the UK has adopted this recommendation, which would carry a considerable cost implication. The evidence that the use of these estimates would, in fact, reduce mis-diagnosis compared to the use of the CKD-EPI equation, is not currently convincing.
Interpretation of measurements of serum creatinine and eGFR

Clinicians should be aware that serum creatinine concentration is determined by the balance between creatinine generation (from muscle turnover, but also from meat intake) and creatinine removal (by glomerular filtration, with some tubular secretion).

- Meat intake and intense exercise can cause a temporary rise in serum creatinine concentration without any change in kidney function; ideally, patients should avoid meat ingestion and muscular exercise for 12 h prior to blood sampling for serum creatinine.

- Trimethoprim and cimetidine inhibit tubular secretion of creatinine. Particularly in patients with CKD G3b-5 (in whom tubular secretion contributes a higher proportion of creatinine removal), these drugs can cause a temporary rise in serum creatinine concentration, lasting at least a week, which does not necessarily reflect a change in kidney function.

- The MDRD and CKD-EPI formulae used for eGFR involve an estimation of creatinine generation rate (in proportion to body surface area) from age, sex, and ethnic origin, and allow estimation of GFR in proportion to body surface area (in ml/min/1.73m\(^2\)). Since bigger people have higher metabolic demands than smaller people, this is the best way to express how well a given patient’s kidneys are working.

- Patients with disproportionately high muscle mass for their age and sex (e.g. body builders) will generate more creatinine than predicted, resulting in eGFR values lower than true GFR; patients with disproportionately lower muscle mass (e.g. amputees, patients with spinal cord injury) will generate less creatinine than predicted, resulting in eGFR values higher than true eGFR. Within an individual, changes in eGFR remain a good guide to changes in kidney function.

- The Cockcroft/Gault formula relies on age, weight and sex, and allows estimation of absolute GFR (i.e. not normalised to body size). It can give more accurate estimates of absolute GFR in people with very high muscle bulk, but its reliance on weight as one predictor of creatinine generation makes it unreliable in obese people. For historical reasons, the BNF recommends the use of this formula for drug dosage adjustment for many drugs. In the great majority of patients, it is reasonable to use the eGFRcreatinine values when altering drug dosage. In patients at the extremes of body size, neither formula is reliable.

- If true GFR needs to be known, it should be measured – either using 24h creatinine clearance (if a complete and accurately timed 24h urine can be collected, combined with a concurrent serum creatinine measurement) or by isotope GFR measurement.
Monitoring and referral of patients with CKD to Nephrology

At least 10% of the UK population meet the current criteria for CKD. Only a small minority will gain added value from specialist management. The NICE referral criteria are designed to ensure that patients with potentially treatable specific causes of CKD, those with progressive CKD, those likely to require treatment for complications of CKD, and those likely to require renal replacement therapy, are all referred in a timely fashion. The majority of patients with CKD require regular monitoring in primary care. The frequency of monitoring depends on the stage of CKD (see figure overleaf).

NICE CG182 recommends referral of patients fulfilling the following criteria:

- GFR < 30 ml/min/1.73m² (CKD stage G4 or G5)
- ACR ≥ 70 mg/mmol, unless known to be caused by diabetes and already appropriately treated (e.g. already on the maximal tolerated dose of an ACEI or ARB)
- ACR ≥ 30 mg/mmol with haematuria
- Sustained decrease in eGFR of 25% or more, and a change in GFR stage
- Sustained decrease in eGFR of ≥ 15 ml/min/1.73/m² within 12 months

Referral of patients with diabetes

Previous NICE guidelines (CG87) suggested that diabetic patients with raised urine albumin excretion but without evidence of retinopathy should be considered for referral to Nephrology. This conflicted with CG182. CG87 has now been superseded by NG28, which states:

Diabetic kidney disease

1.7.12 For guidance on managing kidney disease in adults with type 2 diabetes, see the NICE guideline on chronic kidney disease in adults.

While it is true that diabetic kidney disease is usually associated with retinopathy (the absence of which should therefore prompt consideration of non-diabetic kidney disease), the diagnosis of non-diabetic kidney disease with an ACR < 70 mg/mmol is unlikely to change management. So, patients with diabetes and ACR up to 70 mg/mmol who do not meet one of the eGFR-based criteria for referral should be managed in primary care (with ACEI/ARB-based therapy for blood pressure), irrespective of the presence of absence of retinopathy.

Referral of patients with polycystic kidney disease

Autosomal dominant polycystic kidney disease is a major cause of progressive CKD and end-stage kidney failure. Until recently, no effective treatment was available. Recently, NICE approved tolvaptan (an expensive drug, also licensed for SIADH) for use in patients with CKD stage 2 or 3 due to ADPKD with rapid progression of CKD. All patients with suspected or proven ADPKD should be seen at least once in the Nephrology clinic to receive counselling, including genetic counselling, and to reach a decision on whether they meet the NICE criteria to be offered tolvaptan.

Detection of structural abnormalities indicating CKD

The NICE definition of CKD includes structural abnormalities of the kidneys. These are most commonly detected by ultrasound scanning. However, ultrasound may also show minor structural abnormalities of
the kidney that do not indicate serious kidney disease. For the purposes of this guideline, patients with the following abnormalities should be considered to have chronic kidney disease:

- polycystic kidney disease (multiple cysts with distortion of normal renal anatomy and increase in overall kidney size): NB all patients should be referred to Nephrology
- polar scars suggesting reflux nephropathy
- increased cortical echogenicity
- decreased cortico-medullary differentiation
- decreased cortical thickness

**Renal Cysts**

Patients with simple renal cysts should not be referred to Nephrology unless there is a family history of cystic kidney disease, in which case the patient should be referred to the Renal Genetics Clinic. If the ultrasound report or clinical situation raises the possibility of renal tract cancer (for instance, septated or calcified cysts), the patient should be referred to Urology (for further information, refer to North of Tyne and Gateshead Guidelines for Management of Common Urological Conditions – see page 8). If the ultrasound report suggests polycystic kidney disease, refer to the Renal Genetics Clinic.

**Angiomyolipomas**

Angiomyolipomas are commonly reported as incidental findings on urinary tract ultrasound. They are benign tumours comprising fatty tissue and thick-walled blood vessels. They can occur in association with Tuberous Sclerosis and with Lymphangioleiomyomatosis: in these conditions they are usually multiple and large.

Large AMLs can develop aneurysms, which then bleed, causing retroperitoneal haemorrhage or visible haematuria, with flank pain. The risk of bleeding depends on the size of the AML, and is very low if the diameter is < 4 cm.

Patients without a diagnosis of Tuberous sclerosis or lymphangioleiomyomatosis, with a single lesion < 2 cm, that has the typical appearances of an AML, do not require referral. They should undergo repeat ultrasound at one year and two years. If there is no increase in size during this time, no further imaging is required. If the lesion increases in size then refer to Urology.

Patients with a single AML > 2 cm, or multiple AMLs, should be referred to Urology for CT scan and assessment. Patients with multiple AMLs should also be referred to the Renal Genetics Clinic to exclude tuberous sclerosis.

AMLs can grow more rapidly during pregnancy. Patients with an AML (even if single and < 2 cm) who are pregnant or plan pregnancy should be referred to a Urologist and to a Medical Obstetric Clinic.

**Hydronephrosis**

Hydronephrosis indicates a problem with drainage of urine from the kidney, and should prompt urgent referral to Urology. The patient should be simultaneously referred to Nephrology only if they fulfil other referral criteria.

**Referral for hypertension**

NICE guidelines suggest referral for resistant hypertension (defined as clinic-measured BP > 140/90 mmHg despite maximal tolerated doses of a 3-drug combination according to the AB/CD algorithm); and for early onset hypertension (e.g. age < 40 y).

**Referral for metabolic investigation of kidney stones**

Up to 75% of patients who form a symptomatic kidney stone eventually form a second stone, but this may take 25 years. Patients are unlikely to make major changes to their lifestyle or to take drug...
treatment to prevent stone recurrence unless they have frequent symptomatic stone disease. The referral criteria below are designed to ensure that patients with specific, treatable causes of recurrent kidney stone formation are referred for metabolic investigation. Most such referrals will come from Urology. Patients meeting the following criteria should be offered a Nephrology referral (see the flow chart overleaf):

- Pure calcium phosphate stone
- Cystine stone or elevated cystine excretion on urine biochemistry
- Previous gastric bypass or short gut syndrome
- Strong family history of stone formation
- Nephrocalcinosis confirmed on CT scan
- Formation of kidney stones in childhood
- Hypercalciuria (24h urine calcium >8 mmol/24h despite advice on low salt diet)
- Hyperoxaluria (24h urine oxalate > 0.5 mmol/24h)
- Hypocitraturia (24h urine citrate < 0.7 mmol/24h) on a 24h urine sample taken in the absence of urinary tract infection

See Flow chart overleaf
Kidney Stone Patient Metabolic Assessment

1st presentation

Serum Urate
- Raised
  - Dietary advice (reduce meat), GP to consider allopurinol

Serum Ca^{2+}
- Raised
  - Refer to Endocrinology

Spot Urine Ca^{2+}: creatinine
- Raised
  - Normal - Advise high fluid intake, low salt diet, minimise cola intake

24 hour urine Ca^{2+}, citrate, oxalate, cystine
- Normal
  - Isolated mild hypercalciuria (up to 8mmol/24 h)
- Abnormal
  - Advice: increase fluid intake (reduce Na\(^+\) in diet (reduce processed food + don’t add salt), + check PTH, vit D + repeat 24 hour urine after diet modification

Multiple (2-3) stone episodes or presentation <25

Pure calcium phosphate stone (?RTA)

Cystine stone or elevated urine cystine

Previous gastric bypass (?enteric hyperoxaluria)

Nephrocalcinosis

Strong Family History

Childhood stones

Dietary advice (reduce meat), GP to consider allopurinol

Refer to Endocrinology

Normal - Advise high fluid intake, low salt diet, minimise cola intake

Isolated mild hypercalciuria (up to 8mmol/24 h)

Advice: increase fluid intake (reduce Na\(^+\) in diet (reduce processed food + don’t add salt), + check PTH, vit D + repeat 24 hour urine after diet modification

Abnormal
Resources for patients

**Patient leaflet for people who have had Acute Kidney Injury**, published by the British Kidney Patients Association and the Royal College of General Practitioners

**Patient leaflet for people at risk of Acute Kidney Injury** (including those with Chronic Kidney Disease), published by the British Kidney Patients Association and the Royal College of General Practitioners

**Patient leaflet on Chronic Kidney Disease**, published by Kidney Care UK.

**Patient leaflet on Chronic Kidney Disease**, published by the National Kidney Federation

**Web page on Chronic Kidney Disease**, published by Kidney Research UK

**Information booklet on looking after your kidneys**, published by Kidney Research UK

**Dialysis decision aid**, published by Kidney Research UK – comprehensive written information on dialysis options

**Patient decision aid: established kidney failure**, published by NHS Right Care – web-based, requires registration, helps patients to decide between dialysis and conservative care

**Patient decision aid: dialysis**, published by NHS Right Care – web-based, requires registration, helps patients to decide which modality of dialysis would be best for them

**Patient decision aid: kidney transplant**, published by NHS Right Care – web-based, requires registration, helps patients to decide between transplants from living kidney donors and deceased donors

**Low potassium diet advice**, from The National Kidney Federation