

Classification of CKD based on eGFR and ACR			ACR		
			ACR <3	ACR 3–30	ACR >30
			A1	A2	A3
eGFR	≥90	G1	No CKD IF no other signs of kidney damage		
	60–89	G2			
	45–59	G3a	See * below		
	30–44	G3b			
	15–29	G4			
	<15	G5			

\* If eGFR persistently 45–59 over at least 90d AND ACR <3, do eGFR<sub>cysC</sub>, if available: if eGFR<sub>cysC</sub> ≥60 then they do NOT have CKD. NICE do not say what to do if eGFR<sub>cysC</sub> is NOT available – presumably they have CKD stage G3aA1.

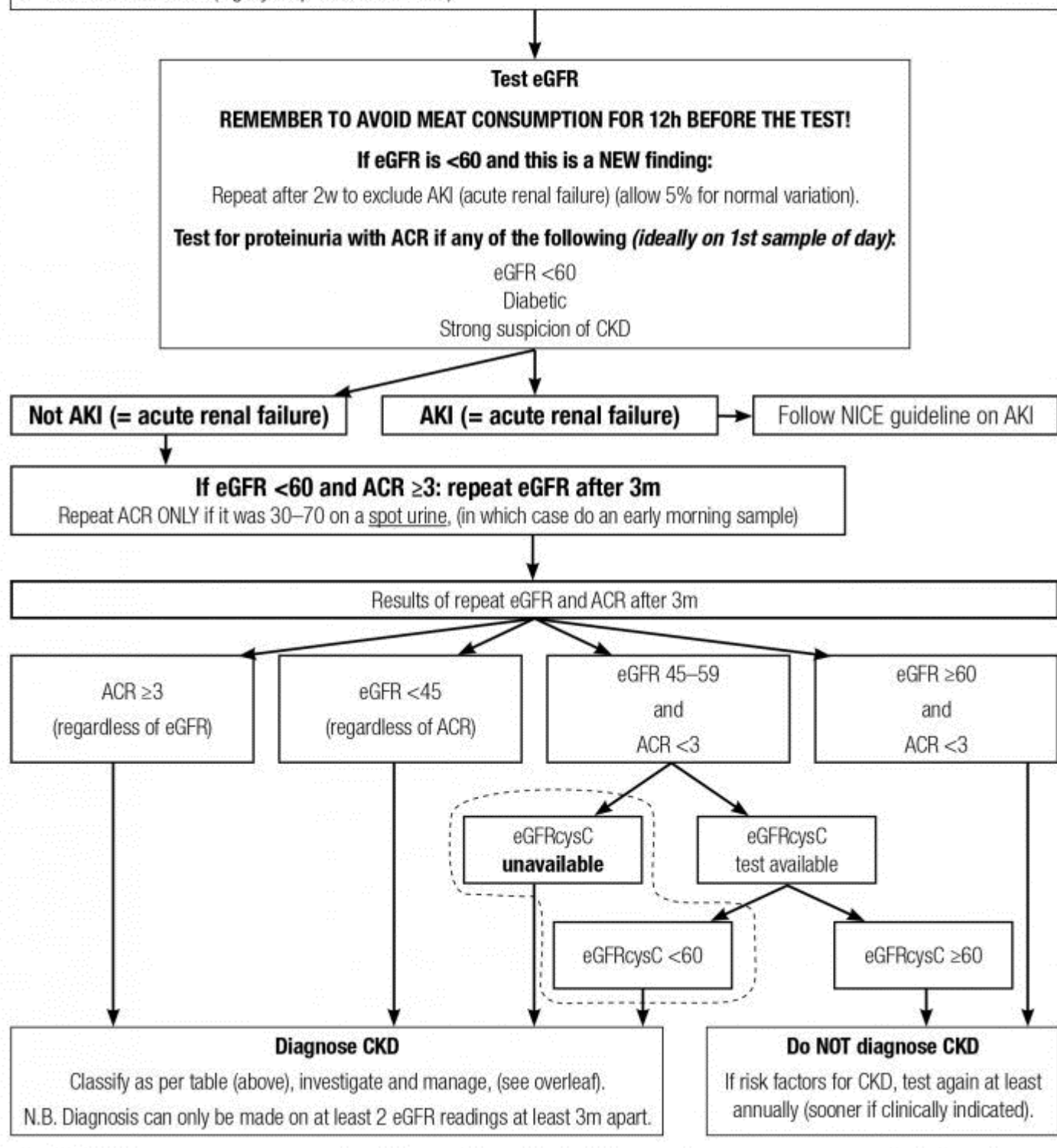
### Offer CKD testing to those with:

- Diabetes.
- Hypertension.
- Cardiovascular disease (IHD, chronic heart failure, cerebrovascular disease and peripheral arterial disease).
- Renal disease (structural renal tract disease, recurrent renal calculi, acute kidney injury (monitor for at least 2–3y after AKI)).
- Prostatic hypertrophy.
- Opportunistically detected haematuria (I presume they mean haematuria alone not, for example, with a UTI).
- Multisystem disease with potential kidney involvement, e.g. SLE.
- Family history of end-stage renal disease (eGFR <15) or hereditary kidney disease.

### Offer at least annual eGFR testing for those on nephrotoxic drugs (based on consensus opinion)

NICE don't give an exhaustive list of nephrotoxic drugs, but do mention the following (and I'd add ACE inhibitors/ARBs):

- Long term NSAID users ('long term' is not quantified) (BNF says monitor those on NSAIDs only if renal impairment).
- Lithium.
- Calcineurin inhibitors (e.g. cyclosporin or tacrolimus).



At GP Update we are not convinced of the benefits of diagnosing CKD in the individuals highlighted by the dashed circle – for the reasons outlined at the start of this article.

CKD diagnosed if:		
eGFR <60 on at least 2 occasions at least 3m apart OR any eGFR if ACR ≥3		
Investigations:		
Bloods	Dip urine for haematuria if ACR ≥3	Renal ultrasound scan
<ul style="list-style-type: none"> <li>If eGFR &lt;45: Hb.</li> <li>If eGFR &lt;30: calcium, phosphate, PTH and vitamin D.</li> </ul>	<ul style="list-style-type: none"> <li><b>Persistent microscopic haematuria (1+ or more on at least 2 out of 3 occasions):</b> rule out cancer if ≥50y.</li> <li><b>Persistent microscopic haematuria in the absence of proteinuria (and cancer):</b> annual follow-up (eGFR, ACR, BP and dipstick for haematuria).</li> </ul>	<ul style="list-style-type: none"> <li>eGFR &lt;30.</li> <li>Progressive CKD (defined below).</li> <li>Haematuria: macroscopic or persistent microscopic haematuria.</li> <li>Symptoms of urinary outflow tract obstruction (hesitancy, straining, poor stream, terminal dribble).</li> <li>Family history of polycystic kidney disease (and over 20y).</li> </ul>
Consider referral if:		
eGFR	ACR	Other
<ul style="list-style-type: none"> <li>eGFR &lt;30.</li> <li>Progressive CKD: <ul style="list-style-type: none"> <li>sustained fall in eGFR of ≥25% AND change in eGFR category in &lt;12m</li> <li>OR</li> <li>sustained fall in eGFR of 15 in &lt;12m</li> </ul> </li> </ul>	<ul style="list-style-type: none"> <li>ACR ≥30 AND haematuria.</li> <li>ACR ≥70 UNLESS caused by diabetes and on appropriate treatment (ACE inhibitor/ARB).</li> </ul>	<ul style="list-style-type: none"> <li>BP poorly controlled on ≥4 drugs.</li> <li>Suspected renal artery stenosis (BP hard to control).</li> <li>CKD with renal outflow obstruction (refer urology unless urgent renal problem, e.g. uraemia, hyperkalaemia, fluid overload).</li> <li>Suspected rare or genetic cause.</li> </ul>
Management for all		
Education and lifestyle	BP control	Statins
<ul style="list-style-type: none"> <li>Understanding of CKD, what can be done to influence course of disease, pros and cons of treatment, and when relevant, advice about dialysis/transplant).</li> <li>All the usual lifestyle changes (stop smoking, healthy weight, exercise etc.).</li> <li>No special diets except in severe disease (under renal team advice).</li> </ul>	<p><b>Aim for:</b> <b>BP &lt;140/90</b> <b>(target systolic BP 120–139).</b> <i>(QOF target 140/85)</i></p> <p><b>In diabetics with ACR ≥70 aim for:</b> <b>BP &lt;130/80</b> <b>(target systolic BP 120–129).</b> <i>(QOF target in diabetes 140/80)</i></p> <ul style="list-style-type: none"> <li><b>Follow NICE hypertension guideline.</b></li> <li><b>Use ACE inhibitors/ARBs if:</b> <ul style="list-style-type: none"> <li>ACR ≥70</li> <li>ACR ≥30 AND hypertension</li> <li>ACR ≥ 3 AND diabetic.</li> </ul> </li> </ul> <p>(For practical advice on use of ACE/ARB in CKD, see below.)</p>	<p><b>For primary AND secondary prevention of cardiovascular disease:</b></p> <ul style="list-style-type: none"> <li>Offer statins to all with CKD.</li> <li>Use atorvastatin 20mg. <ul style="list-style-type: none"> <li>If eGFR ≥30: increase dose if &lt;40% fall in non-HDL cholesterol.</li> <li>If eGFR &lt;30 increase dose only in consultation with renal team.</li> </ul> </li> <li>Do not assess CVD risk using QRISK2 or other risk assessment tools.</li> </ul>
<p><b>In severe disease: also manage anaemia, bone and metabolic disorders.</b></p> <p>This may include prescribing treatments for anaemia, vitamin D (colecalfiferol, ergocalciferol or alfacalcidol), or sodium bicarbonate if metabolic acidosis present.</p>		
Monitoring		
<p>QOF requires annual monitoring. Tailor monitoring to the individual based on:</p> <ul style="list-style-type: none"> <li><b>Severity of CKD and patient's past history of eGFR variation</b> (if declining rapidly, test often, if stable, test infrequently).</li> <li>Co-morbidities, especially heart failure (people with heart failure are much more sensitive to changes in renal function).</li> <li>Changes to treatment, especially NSAIDs, diuretics and RAAS drugs (ACE inhibitors, ARBs, aliskiren, spironolactone).</li> <li>Inter-current illness.</li> <li>Cause of CKD.</li> <li>If they have chosen conservative management (interestingly NICE don't mention this concept elsewhere in the guidance).</li> </ul>		