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Discharge after acute kidney injury – recognising and managing risk

Abstract

Acute kidney injury (AKI) is common and serious. While it is usually cared for in hospital, it is no less important in primary care. AKI typically arises first in the community and is often not completely resolved at hospital discharge. Primary care has a role both in prevention and follow up of AKI. Surveillance for new chronic kidney disease (CKD) is recommended in guidelines. In this article we compare the diagnostic criteria for AKI and CKD, what a diagnosis means in primary care, and the role of primary care in monitoring for CKD after AKI.

Keywords

(MeSH): acute kidney injury; chronic kidney disease; patient safety; primary care

AKI and CKD – making the diagnosis

Acute kidney injury (AKI) is serious and common. It complicates 1 in 7 hospital admissions. It affects patients in all clinical settings, but typically develops first in the community either before prompting or becoming apparent on hospital admission. A recent National Confidential Enquiry into Patient Outcome and Death (NCEPOD) report was critical of inpatient care in AKI, citing delays in recognition and treatment as causes of preventable harm[1]. The diagnosis of AKI requires an abrupt change in serum creatinine or urine output presumed to have occurred within the past week.

Chronic kidney disease (CKD) is a sustained abnormality of kidney function or structure. Either functional decline (e.g. measured with estimated glomerular filtration rate, eGFR) or structural change (e.g. albuminuria) should be evident for at least three months. Approximately 11.5% of the population meet these criteria, and this prevalence increases greatly with age[2]. Only a minority of patients develop end-stage kidney disease but a much greater proportion suffer from complications including renal bone disease, anaemia, vascular calcification, cardiovascular disease and admissions with AKI. The Kidney Disease: Improving Global Outcomes (KDIGO) criteria for both AKI and CKD are below (Table 1).

AKI and CKD – interpreting the diagnosis

To correctly interpret these seemingly precise criteria, one must understand their limitations and how they are different. They differ not just in time frame, but also in the measures involved. The CKD criteria use eGFR, whereas the AKI criteria use changes in creatinine. eGFR is not an exact measurement of glomerular

Table 1. AKI and CKD diagnostic criteria[3, 4]

KDIGO AKI Definition	KDIGO Stage	Creatinine Change	Urine Output
Increase in serum creatinine of ≥26.4μmol/l within 48 hours or ≥1.5 fold rise in creatinine presumed within 7 days or urine output <0.5ml/kg/hr for 6 hours	1	≥26.5μmol/l or >1.5 fold rise	<0.5mg/kg/hr for 6 hours
	2	>2 fold rise	<0.5mg/kg/hr for 12 hours
	3	>3 fold rise or creatinine≥353.6μmol/l having first met the definition or Acute RRT	<0.3mg/kg/hr for 24 hours or anuria for 12 hours
KDIGO CKD Definition	**KDIGO Category**	**Functional or Structural Derangement**	
Evidence for > 3 months of at least one of: KDIGO category G3a or A2 *	G1	eGFR≥ 90 ml/min/1.73m² (normal/high)*	
	G2	eGFR 60-89 ml/min/1.73m² (mild decrease)*	
	G3a	eGFR 45-59 ml/min/1.73m² (mild/moderate decrease)	
	G3b	eGFR 30-44 ml/min/1.73m² (moderate/severe decrease)	
	G4	eGFR 15-29 ml/min/1.73m² (severe decrease)	
	G5	eGFR <15 ml/min/1.73m² (kidney failure)	
	A1	Urinary ACR <3mg/mmol (normal/mild increase)*	
	A2	Urinary ACR 3-30mg/mmol (moderate increase)	
	A3	Urinary ACR >30 mg/mmol (severe increase)	

*CKD can also be defined by abnormal urine sediment; electrolyte abnormality due to tubular disorder; abnormality detected by histology or imaging. eGFR = estimated glomerular filtration rate; ACR = albumin creatinine ratio

filtration, but an *estimation* derived from serum creatinine. The estimate is based on the assumption that creatinine production from muscle and elimination in urine are in a steady state. In AKI this is not the case.[5]

For instance, a hypothetical patient who has surgical removal of 1½ kidneys clearly has a sudden reduction of *true* GFR, but this will not be immediately reflected in the blood results. Just as a sink takes time to fill with water when the drain is plugged, serum creatinine will spend several days rising steadily until a new balance develops between creatinine production (the water tap) and elimination (the blocked drain). Estimated GFR if based on a creatinine taken early before this new balance has been achieved will not reflect the true change in GFR that has occurred. In practice, this means that the clinician must look not only at the most recent result, but also review the trend in changes from previous tests, particularly in a patient who is either acutely unwell, or recovering from an acute illness. This is undoubtedly easier if a patient with risk factors for developing AKI is already under regular surveillance (such as a disease register) and has those previous tests available.

AKI and CKD – an early diagnosis may prevent harm

The AKI and CKD criteria identify renal impairment, but do not explain why it has developed or what should be done next. They highlight the patient who is either acutely unwell (AKI) or could be vulnerable if they were to become unwell (CKD), but both the AKI and CKD guidelines stress that the next step should be to consider the underlying cause[3, 4]. The clinician should aim to address any concurrent illness (e.g. sepsis) or modifiable factors (e.g. non-steroidal anti-inflammatory, NSAID use) that might improve kidney function, prevent recurrence, or prevent further deterioration.

This is especially important for AKI in the hospital where the acutely unwell patient can quickly deteriorate if not recognised and sometimes a rising creatinine is the first signal that a patient is unwell. The same recommendations, however, also apply in the community. Most AKI develops first in the community, and patients who recover from AKI will eventually be discharged back into the community. This means that primary care has an important role in the primary prevention of AKI, early recognition of AKI when it occurs, and prevention of recurrence after patients return home.

However, preventing and recognising AKI is only possible when a clinician is aware of who is at risk and checks a blood test when appropriate. Helpfully, the risk factors for AKI and CKD progression reported in the National Institute for Health and Clinical Excellence (NICE) clinical guidelines (CG169, CG182) (Table 2) are strikingly similar[6, 7]. Both CKD and its risk factors substantially increase the risk of developing AKI. These are often patients with multi-morbidity and less physiological reserve who are already likely to be on a disease register as part of the Quality Outcomes Framework (QOF)[8]. A patient on a coronary heart disease, heart failure, stroke, diabetes, dementia, learning disability or CKD register, who presents to your clinic with an acute illness is a patient who may also be developing AKI.

Recognising risk after AKI – an uncertain period

In addition to diagnosis, another role of primary care is in the aftermath after AKI has occurred, as patients

Table 2. Risk factors reported in the NICE clinical guidelines for AKI (CG169) and CKD (CG182)[6, 7]

AKI risk factors	CKD progression risk factors
Previous AKI	Previous AKI
Chronic kidney disease	Hypertension and proteinuria
Heart failure	Cardiovascular disease
Diabetes	Diabetes
History of urinary tract obstruction	History of urinary tract obstruction
Recent use of NSAIDs	Chronic use of NSAIDs
Liver Disease	Smoking
Acute illness with sepsis, hypovolemia or oliguria	African, African-Caribbean or Asian origin
Recent drug with nephrotoxic potential (ACE inhibitors, ARBs, aminoglycosides, iodinated contrast)	
Disability limiting access to fluids, or reliance on a carer	
Age over 65	

remain at risk when they return to the community. In the past, AKI was considered a reversible syndrome. If a patient survived it was once assumed that the kidney function would later return to a more or less acceptable level. This is no longer considered the case. Evidence now links AKI with increased mortality, progressive CKD, cardiovascular events and chronic dialysis requirement years after the initial episode[9]. Patients discharged after AKI are also at increased risk of being promptly readmitted[10].

Recovery is not easy to interpret. The changes in kidney function after AKI are often complex and this is why close monitoring is required. In the year after an AKI episode, some patients will continue to improve, some patients will level off at a new baseline level of function and some patients will be vulnerable to further decline. This ongoing recovery or decline is in part related to compensatory changes in the remaining nephrons and changes in persisting risk factors for AKI and CKD.

The reliability of creatinine based markers of kidney function can also vary. For instance, some patients recovering from AKI in critical illness can appear to have better kidney function than before their illness. This is deceptive and often relates to reduced muscle mass and reduced creatinine generation rather than improved glomerular filtration. Once muscle mass is later restored, the creatinine will paradoxically rise (and thus eGFR drop) even if actual kidney function has remained unchanged. Other factors also affect the estimation of kidney function and are summarised in Table 3. If the interpretation of creatinine is unclear (e.g. with extremes of muscle mass), the NICE CKD guidelines suggest

Table 3. Factors affecting the estimation of kidney function[11-13]

Mechanism	Examples
Increase in muscle creatine breakdown product (exogenous or endogenous sources)	Dietary load (meat, supplements)
	Rhabdomyolysis
	Increased muscle mass
Decrease in muscle creatine breakdown product	Vegetarian diet
	Muscle wasting
	Recent critical illness
	Amputation
Increased volume of distribution of creatinine	Large volume fluid resuscitation
	Congestive cardiac failure
	Ascites
Inhibition of proximal tubule secretion of creatinine	Trimethoprim
Interference with creatinine assay	Bilirubin
	Hyperglycaemia
	Cephalosporins
Causes of transient Proteinuria	Febrile illness (e.g. pneumonia)
	Congestive cardiac failure
	Urinary tract infection
	Seizures

GFR estimation using cystatin C for the diagnosis and classification of CKD, but this is not available everywhere and is also not perfect[7].

The weeks after hospital discharge from AKI, are therefore critical. Some patients may have ongoing illness, some may be vulnerable to recurrent insults and some may have modifiable risk factors that have been overlooked. Without a carefully considered plan a patient may promptly be readmitted with recurrent illness, or may represent months later (too late) with advanced renal impairment that could have been avoided.

Recognising risk after AKI - the handover from hospital to the community

Patients with AKI still may be at risk after discharge, and they are also numerous. The majority are looked after by non-nephrologists while in hospital and after discharge very few receive outpatient nephrology follow up. Monitoring of kidney function after discharge should therefore be specifically discussed on discharge with a clear transfer of responsibility to ensure patients do not slip through the net.

Many AKI and CKD patients have medications changed while in hospital. Some drugs will have been stopped with permanent intent, and others with the intention of restarting or reviewing. Some decisions may involve a risk/benefit trade-off involving more than one specialty (e.g. ACE inhibitors in kidney disease and cardiac disease). Polypharmacy in CKD also can make any changes difficult to track.

A clear follow-up plan including medication reconciliation is therefore vital. Unfortunately, the immediate discharge letter is often written by the most junior, most busy, most stressed member of the medical team, who may not have met the patient other than to take their blood. Although some queries can wait until the finalised discharge letter, some complex decisions are best discussed while the patient is still fresh in the memory of all involved. Thus, proactive action or clarification is sometimes required and the sections that follow discuss the current recommendations and main issues that should be covered.

Managing risk after AKI – what is recommended?

Monitoring is not possible unless AKI is first recognised and receives good initial care. This requires an awareness of patients at risk and appropriate preventative measures in hospital and the community. Much work has been done recently to improve awareness and understanding of AKI, in particular through the NHS England "Think Kidneys" campaign (www.thinkkidneys.nhs.uk).

Automated e-alerts to healthcare providers when blood tests suggest that AKI may be present are an encouraging development. Automated AKI detection is already mandatory in NHS England hospitals and introduction is also intended for primary care in 2016. If accompanied with appropriate education this initiative has potential to enhance the avoidance and early recognition of AKI and reduce missed opportunities for timely intervention[14].

In NHS England, the Commissioning for Quality and Innovation (CQUIN) scheme for 2015/16 now includes goals for acute kidney injury. Patients who have had AKI in hospital should have this recognised on their discharge summary, including stage of AKI; evidence of medications review and the type and frequency of blood tests required after discharge[15]. This should enhance the visibility of vulnerable patients returning to the community. It also complements the GP Quality and Outcomes Framework (QOF 2015/16), which encourages the identification of CKD in the community by requiring a register of patients aged 18 and over with CKD stages G3a-5 in line with the classification in table 1[8].

Both national and international guidelines recommend monitoring for CKD after AKI. The KDIGO AKI guidelines advise that all patients should be assessed at 90 days after AKI for evidence of new or worsening kidney function[3]. The NICE CKD guidelines also advise that all patients who have had an episode of AKI should be considered at risk of developing CKD or of CKD progressing to a more advanced stage. They recommend that all patients should be monitored for at least 2-3 years after an AKI episode even if the creatinine has returned back to baseline (pre-illness) level[7]. They also suggest monitoring frequencies of at least six monthly in patients in categories G3b (GFR <45ml/min/1.73m^2) or A3 (albuminuria >30mg/mmol) (and more often in more advanced disease), but acknowledge that these can be tailored to the individual.

Managing risk after AKI – the post discharge review

The overall prognosis of AKI is of increased mortality and CKD progression, but the specific factors that predict persisting poor outcomes are the subject of ongoing clinical research. In addition, AKI encompasses a variety of illness states requiring different management approaches. To assist, some areas provide local guidelines for priorities in the post-discharge review after AKI. These may include items outlined in the "RISK" assessment below.

R - Renal diagnosis and cause

Most AKI is not related to renal parenchymal disease, but occurs in the context of acute severe illness including sepsis, ischaemia, bleeding, volume depletion and hypotension. The cause of AKI should be noted for future reference for two reasons. First, recurrent AKI often follows a similar mechanism. For instance, recurrent AKI after urinary obstruction requiring a ureteric stent may now be related to a blocked stent; or a previous AKI following ACE inhibitor introduction may increase the suspicion that recurrent AKI with pulmonary oedema is due to renal artery stenosis. Second, the development of AKI introduces the possibility of related conditions. For instance a smoker or a patient with diabetes who develops AKI may already have other undiagnosed vascular diseases.

I - Intervenable risk factors

Some AKI and CKD risk factors are recurrent or longstanding and can be mitigated by taking pre-emptive avoiding action. For instance, developing strategies to avoid recurrent dehydration in a patient with a high output stoma; or cautious adjustment of diuretics in a patient with congestive cardiac failure and recurrent AKI.

S - Sick day rules and medications review

Drugs affecting renal blood flow are frequently implicated in AKI. NSAIDs, renin-angiotensin system (RAS) blockers such as ACE inhibitors and diuretics can precipitate and exacerbate pre-renal AKI, particularly when used in combination ("the triple whammy"). NSAIDs should be avoided after AKI and consideration should be given as to whether other drugs are necessary. It would be appropriate to stop antihypertensive agents if the risks of recurrent AKI and hypotension clearly outweigh any long-term protective benefits. In patients with an important indication such as those with heavy proteinuria, it may be advisable to continue RAS blockers and diuretics. "Sick day rules" have recently become popular in many areas in this circumstance, advocating temporary discontinuation in the setting of significant diarrhoea, vomiting or febrile

Table 4. Specialist referral criteria in NICE guidelines for CKD (CG182)[7]

Specialist assessment should be considered for:
GFR < 30ml/min/1.73m^2, with or without diabetes
Albumin creatinine ratio ≥ 70mg/mmol, unless known to be due to diabetes and on appropriate treatment
Albumin creatinine ratio ≥ 30mg/mmol with haematuria
Fall in GFR ≥ 25% with a change in GFR category, or GFR fall ≥ 15 ml/min/1.73m^2 over 12 months
Poorly controlled hypertension despite use of 4 agents at therapeutic doses
Suspected rare or genetic cause of CKD (e.g. primary renal disease)
Suspected renal artery stenosis

illness. While there have been encouraging reports that this may reduce community AKI without unintended consequences, an evidence base is currently outstanding and care may be required in some patients with cardiac failure.

At the time of the post-AKI review some drugs may have already been discontinued temporarily. The re-introduction of antihypertensive agents and diuretics should be accompanied by reassessment of blood pressure and fluid balance. Creatinine and potassium should also be rechecked 1-2 weeks after any RAS blocker dose increase.

K - Kidney recovery or progression

Prompt recovery of kidney function back to baseline (pre-illness) level is associated with better outcomes after AKI[16]. Assessment should include testing both for return of creatinine to baseline, and for persistent proteinuria now that the acute illness has resolved. Monitoring for recovery is vital and may be used to predict and prevent recurrence, readmission and avoidable death. A patient with persisting renal impairment should prompt a reassessment to find out why. It may be a sign of ongoing acute illness, an incorrect diagnosis, an overlooked causative factor, or the development of CKD. The specific CKD referral recommendations from NICE are outlined in Table 4.

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Competing Interests

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Key Points Summary

- Patients with AKI come from the community and return to the community. Primary care therefore has a vital role in recognising AKI and preventing recurrence.
- Care must be taken to interpret creatinine and eGFR correctly in acute illness. The change is often more important than the absolute value.
- Patients with AKI frequently have risk factors covered by disease registers as part of the QOF.
- AKI is a serious illness, but patients often return to the community before recovery is complete.
- Patients with AKI have a sustained increased risk of relapse, recurrence, CKD progression and death.
- A good handover on discharge to primary care is essential as is included in the national CQUIN.
- Specific management after AKI is individual to the patient, but may involve:
  1. R - confirm the renal diagnosis
  2. I - address any intervenable risk factors to prevent or pre-empt recurrence
  3. S - sick day rules and medications review
  4. K - assess for kidney recovery or the development of new CKD

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Discharge after acute kidney injury - recognising and managing risk

1. A 76 year old man with a four day history of vomiting and profuse diarrhoea is reviewed at home. Past medical history includes hypertension and osteoarthritis of the knees. Medication - amlodipine, dihydrocodeine, candesartan, naproxen. On examination he is pyrexial, drowsy, with reduced skin turgor and dry mucous membranes. Blood pressure 96/62, HR 112 regular. Chest is clear. He has mild abdominal tenderness without guarding. Blood tests include potassium 5.4, albumin 29, urea 27, creatinine 238, eGFR from lab 20. Urinalysis protein++ .His last creatinine was 120 more than three years ago.

How would you classify this man's renal impairment? Select only one option.

a. Acute kidney injury
b. Chronic kidney disease
c. End stage kidney disease (kidney failure)
d. Insufficient information
e. Normal kidney function

2. A 76 year old man with a four day history of vomiting and profuse diarrhoea is reviewed at home. Past medical history includes hypertension and osteoarthritis of the knees. Medication - amlodipine, dihydrocodeine, candesartan, naproxen. On examination he is pyrexial, drowsy, with reduced skin turgor and dry mucous membranes. Blood pressure 96/62, HR 112 regular. Chest is clear. He has mild abdominal tenderness without guarding. Blood tests include potassium 5.4, albumin 29, urea 27, creatinine 238, eGFR from lab 20. Urinalysis protein++ .His last creatinine was 120 more than three years ago

Which of the following best describes the underlying renal diagnosis? Select only one option.

a. Hypertensive nephropathy
b. Nephrotic syndrome
c. Pre-renal illness
d. Renal artery stenosis
e. Obstructive uropathy

3. A 76 year old man with a four day history of vomiting and profuse diarrhoea is reviewed at home. Past medical history includes hypertension and osteoarthritis of the knees. Medication - amlodipine, dihydrocodeine, candesartan, naproxen. On examination he is pyrexial, drowsy, with reduced skin turgor and dry mucous membranes. Blood pressure 96/62, HR 112 regular. Chest is clear. He has mild abdominal tenderness without guarding. Blood tests include potassium 5.4, albumin 29, urea 27, creatinine 238, eGFR from lab 20. Urinalysis protein++ .His last creatinine was 120 more than three years ago

Which of his medications should be discontinued at present? Select only one option

a. Candesartan
b. Candesartan and Naproxen
c. Dihyrocodeine and Naproxen
d. Amlodipine, Candesartan and Naproxen
e. All his medications should be discontinued

4. A 78 year old man is discharged from the urology ward and attends your clinic for review the following fortnight. He had been admitted with kidney stones and was found to have ureteric obstruction. A nephrostomy was inserted, a ureteric stent was passed and the nephrostomy subsequently removed. His previous baseline creatinine was 96 (lab reported eGFR 70), this rose to 194 during admission (lab reported eGFR 31). His last test before discharge showed a creatinine of 136 (lab reported eGFR 47). Urinary dipstick negative.

How would you classify this man's renal impairment? Select only one option.

a. Chronic kidney disease G3a A1
b. Chronic kidney disease G3b A1
c. End stage kidney disease (kidney failure)
d. Insufficient information
e. Normal kidney function

5. **A 78 year old man is discharged from the urology ward and attends your clinic for review the following fortnight. He had been admitted with kidney stones and was found to have ureteric obstruction. A nephrostomy was inserted, a ureteric stent was passed and the nephrostomy subsequently removed. His previous baseline creatinine was 96 (lab reported eGFR 70), this rose to 194 during admission (lab reported eGFR 31). His last test before discharge showed a creatinine of 136 (lab reported eGFR 47). Urinary dipstick negative.**

   Prior to admission he was on furosemide 40 mg once daily, ramipril 10 mg once daily and ibuprofen 400 mg three times daily. These were discontinued while in hospital. ALL of the following statements are False, except one. Select the SINGLE true statement.

   a. All his medications can now be safely restarted.
   b. All his medications can be restarted if his repeat creatinine has returned to baseline.
   c. He should restart his medications one at a time if his creatinine has returned to baseline.
   d. The indications for each medication should be reviewed in conjunction with clinical and laboratory assessment
   e. He should remain off these medications as they were not prescribed on his discharge letter.

6. **A 78 year old man is discharged from the urology ward and attends your clinic for review the following fortnight. He had been admitted with kidney stones and was found to have ureteric obstruction. A nephrostomy was inserted, a ureteric stent was passed and the nephrostomy subsequently removed. His previous baseline creatinine was 96 (lab reported eGFR 70), this rose to 194 during admission (lab reported eGFR 31). His last test before discharge showed a creatinine of 136 (lab reported eGFR 47). Urinary dipstick negative.**

   His repeat creatinine is 186 (lab reported eGFR 33). He is otherwise asymptomatic. What is the next most appropriate course of action? Select only one option

   a. Contact the urology ward to discuss readmission
   b. Maintain regular follow up every 4 – 6 months
   c. Repeat blood test again one week
   d. Refer him for an outpatient urinary tract ultrasound
   e. Refer him to the nephrology outpatient clinic for regular follow up

7. **A 78 year old man is discharged from the urology ward and attends your clinic for review the following fortnight. He had been admitted with kidney stones and was found to have ureteric obstruction. A nephrostomy was inserted, a ureteric stent was passed and the nephrostomy subsequently removed. His previous baseline creatinine was 96 (lab reported eGFR 70), this rose to 194 during admission (lab reported eGFR 31). His last test before discharge showed a creatinine of 136 (lab reported eGFR 47). Urinary dipstick negative.**

   His repeat creatinine is 112. From correspondence you note that he has a history of moderate left ventricular systolic dysfunction and that ramipril was recommended by cardiology to be recommenced on discharge. Which is the most appropriate course of action? Select only ONE option.

   a. Restart ramipril at full dose and review in 3 months.
   b. Monitor blood tests weekly and restart only if creatinine falls below baseline.
   c. Restart ramipril if blood tests at 90 days suggest new onset CKD.
   d. Restart ramipril at a reduced dose and recheck blood tests in two days.
   e. Restart ramipril at a reduced dose and recheck blood test in 1-2 weeks.

8. **A 58 year old woman is discharged from hospital after a six week admission for septic shock including a period of renal replacement therapy for severe AKI, and mechanical ventilation in intensive care. She is on no medication except for nutritional supplements and currently has a BMI of 15. Her creatinine on discharge was 65 (lab reported eGFR 86) and her urinalysis is negative.**

   Which of the following is MOST likely to be true about her current kidney function? Select only ONE option.

   a. Her kidney function has returned to normal
   b. Kidney function will be underestimated due to dietary supplements
   c. Kidney function will be overestimated due to reduced muscle mass
   d. Kidney function will be underestimated due to reduced muscle mass
   e. She has evidence of chronic kidney disease

9. **A 58 year old woman is discharged from hospital after a six week admission for septic shock including a period of renal replacement therapy for severe AKI, and mechanical ventilation in intensive care. She is on no medication except for nutritional supplements and currently has a BMI of 15. Her creatinine on discharge was 65 (lab reported eGFR 86) and her urinalysis is negative. What further monitoring is recommended for this patient? Select only ONE option.**

a. As her kidney function has returned to baseline no formal monitoring is required.
b. She should have urine and creatinine checked at 3 months to check for new CKD. If normal then no further monitoring required.
c. She should have urine and creatinine checked at 3 months to assess for new CKD. She should be monitored for at least 2-3 years regardless of the result.
d. She should have her urine and creatinine checked in 2-3 years.
e. She should be monitored until her BMI has returned to normal.

10. **A 58 year old woman is discharged from hospital after a six week admission for septic shock including a period of renal replacement therapy for severe AKI, and mechanical ventilation in intensive care. She is on no medication except for nutritional supplements and currently has a BMI of 15. Her creatinine on discharge was 65 (lab reported eGFR 86) and her urinalysis is negative. 4 months later her BMI has risen to 19 and her urinalysis remains negative. Her creatinine is now 94 (lab reported eGFR 56). What is your assessment of her kidney function now? Select only ONE option.**

a. She has new onset chronic kidney disease.
b. She may have new onset chronic kidney disease but further assessment is necessary.
c. She has normal kidney function.
d. She has recurrent acute kidney injury stage 1
e. She has recurrent acute kidney injury stage 3

EXTENDED MATCHING QUESTIONS

EMQ 1

THEME: FACTORS AFFECTING ESTIMATION OF KIDNEY FUNCTION

Options:

a. Increase in creatine breakdown product
b. Decrease in creatine breakdown product
c. Increased volume of distribution of creatinine
d. Inhibition of proximal tubule secretion of creatinine
e. Interference with creatinine assay
f. Assumption of a steady state for GFR estimation
g. Transient abnormality in the context of underlying illness

In each of the case scenarios a factor has affected the interpretation of kidney function tests. Match the scenario to the best explanation of the list of factors in the options given, for the blood results and clinical presentation. Each option may be used once, more than once or not at all.

1. **A 65 year old woman with recurrent urinary tract infections is placed on an extended course of trimethoprim. Creatinine rises to 102 from 67 the previous week.**

2. **An 84 year old lady is admitted obtunded with urinary sepsis. She is found to have AKI with a creatinine of 238 from normal baseline and is oliguric. She receives large volume fluid resuscitation but remains oliguric although the creatinine does not rise further.**

3. **A 60 year old receives a bilateral nephrectomy for renal carcinoma. 1 hour post-op he is anuric, but his creatinine is 102 (lab reported eGFR 51). He is scheduled for dialysis the following day.**

4. **A 62 year old man is admitted to hospital with sepsis and severe pneumonia. It is noted on admission that he has protein in his urine and this is sent off for quantification. His ACR (albumin/creatinine ratio) is 18 and serum creatinine 84 (lab reported eGFR 85). The following week his ACR is <3, serum creatinine 88 (lab reported eGFR 81).**

5. **A 64 year old diabetic man with diabetic nephropathy and a baseline creatinine of 240 presents with an ischaemic limb. Revascularisation is ineffective and an above knee amputation is performed. At discharge it is noted that his creatinine has fallen to 200.**

EMQ 2

THEME: RISK FACTORS AND COMPLICATIONS IN KIDNEY DISEASE

Options:

a. Vascular calcification
b. Hypotension
c. Proteinuria
d. Hypoglycaemia
e. Hyperglycaemia
f. Liver Disease
g. Diabetes Mellitus
h. NSAIDs
i. Beta blockers
j. ACE inhibitors

Each clinical scenario describes a risk factor or complication of kidney disease. Match the scenario to the SINGLE factor from the list of options that best fits the description. Each option may be used once, more than once or not at all.

1. **A patient with CKD on ramipril and furosemide in the community is at particularly increased risk of AKI when co-prescribed this type of medication.**

2. **This bedside test finding is associated with CKD progression in the long-term, but can also be transiently abnormal in the context of acute febrile illness.**

3. **This drug is useful in the management of CKD with proteinuria in the community.**

4. **This is an important long term complication of CKD.**

5. **Clinicians may need to adjust medications to prevent this complication from occurring in people with type 2 diabetes mellitus who develop severe acute kidney injury.**

EMQ 3

THEME: ASSESSMENT OF KIDNEY FUNCTION

Options:

a. Serum creatinine
b. eGFR
c. albumin creatinine ratio
d. urinalysis
e. ultrasound scan
f. blood pressure
g. urine output
h. ECG
i. histology

For each scenario given describes a diagnostic test used to assess kidney structure or function. Select, from the options given, the test being described.

1. **This test assumes a steady state that may not exist in an acute setting.**

2. **When available, knowledge of this is may provide the first measureable change suggestive of acute kidney injury.**

3. **If found to be abnormal in a patient who is not recovering during follow up after AKI, this may prompt you to consider an intrinsic renal abnormality.**

4. **This may be falsely low in the recovery phase of critical illness.**

5. **If not already done, this should be considered when a patient is failing to recover from AKI.**

