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Acute kidney injury

Acute kidney injury is a clinical syndrome that has become increasingly recognised as a marker of severity of illness in the acutely sick patient. It can lead to, or worsen, chronic kidney disease, and as a result, increase cardiovascular risk. This article sets out the relevance of acute kidney injury for general practice, detailing the identification, management, follow-up and strategies for prevention.

The GP curriculum and acute kidney injury

Clinical module 3.03: Care of acutely ill people states a GP should be able to:

- Recognise the signs of illnesses and conditions that require urgent intervention
- Work effectively in teams and co-ordinate care
- Prioritise problems and establish a differential diagnosis
- Make the patient's safety a priority
- Accept responsibility for your actions, at the same time recognising any need for involving more experienced personnel
- Act calmly in emergency situations and follow agreed protocols
- Know the processes and arrangements for commissioning and delivering urgent and unscheduled care in your community
- Be aware of how the management of patients with continuing conditions affects the need to give urgent and unscheduled care

What is acute kidney injury?

Acute kidney injury (AKI), previously known as acute renal failure (ARF), is a rapid worsening of renal function caused by an insult to the kidneys, which inhibits fluid, electrolyte and acid/base balance (Lewington and Kanagasundaram, 2011). The term AKI was changed from ARF in 2011 by the Kidney Disease Improving Global Outcome (KDIGO) group to allow a broader spectrum of kidney insult to be included in this umbrella term. AKI is a spectrum of severity, ranging from mild injury to severe impairment. At the most severe end, these patients may require renal replacement therapy.

AKI is a clinical syndrome with multiple aetiologies. The risk of AKI occurring is based on the interaction between the susceptibility of the patient and extent of the exposure to the insult (KDIGO, 2012). AKI is an independent marker of illness, and highlights an acutely sick patient who requires prompt management and treatment.

The diagnosis of AKI is based on acute changes in serum creatinine and urine output. It is divided into stages 1, 2 and 3 based on an acute deterioration of creatinine from the patient's baseline or change in urine output (Table 1). In primary care, AKI is most likely to be diagnosed from changes in creatinine alone, as urine output is very rarely measurable.

AKI can also be defined as a greater than 50% rise in creatinine known or presumed within the last 7 days. Within the primary care setting, the gold standard for diagnosing AKI is reviewing current and previous blood results in the clinical context of the patient. These blood tests should then be compared with the AKI diagnostic and staging criteria.

AKI is clinical syndrome; however, there is an important link with chronic kidney disease (CKD). CKD is a risk factor for AKI, and AKI is a risk factor for developing CKD.

Importance of AKI

AKI is common, harmful, costly but preventable. It is seen in 13–18% of all patients (approximately one in five) admitted to hospital, with a higher prevalence in older adults. (Wang, Munter, Chertow, and Warnock, 2012) There are 100 000

Table 1. Definition of AKI.

AKI stage	Serum creatinine criteria (within 48 hours)	Urine output criteria
1	Increase of creatinine of greater than 26 micromol/L above baseline OR An increase of greater than or equal to 1.5–2 fold from baseline	Less than 0.5 mg/kg/hour for at least 6 hours
2	An increase of greater than or equal to two- to three-fold from baseline	Less than 0.5 mg/kg/hour for at least 12 hours
3	A three-fold increase from baseline OR Serum creatinine of greater than or equal to 355 micromol/L with an acute rise of at least 45 micromol/L	Less than 0.3 mg/kg/hour for at least 24 hours OR Anuria for greater than 12 hours

Source: KIDGO (2011); NICE (2013); Selby et al. (2012).

deaths in hospital from AKI annually, and of these 35% are preventable (Steward, Smith, Kelly, & Mason, 2009). Of all AKI, 65% starts in the community (Selby et al., 2012). However, of this 65%, not all will be preventable.

Costs to the NHS of AKI, excluding community costs, are estimated to be between £434 000 000 and £620 000 000 per year. AKI is more expensive than the combined costs associated with breast cancer, lung cancer and skin cancer (National Institute for Health and Care Excellence (NICE), 2013).

Developing AKI is also a marker for poorer outcomes. The mortality for all AKI is 20% during hospital admission. In AKI stage 3 the mortality rises to more than 35% (Selby et al., 2012). As well as increased mortality risk, AKI is associated with longer length of hospital stay, including increased access to healthcare resources such as intensive treatment units and renal replacement therapy.

There is often incomplete recovery of renal function, and patients are left either with CKD or at an increased risk of progressive loss of renal function over time. This all equates to poorer long-term health outcomes, increased cardiovascular risk (due to the association with CKD and cardiovascular risk), reduced life expectancy and poorer quality of life (KDIGO, 2012; Wonnacott, Meran, Amphlett, Talabai, and Phillips, 2014).

Causes of AKI

There are many causes of AKI; the most common ones are hypovolaemia, hypotension, sepsis and medications (KDIGO, 2012). AKI often occurs in the context of other serious illness, which can be viewed as AKI risk factors (Box 1).

Box 1. Patient risk factors for AKI.

- CKD: Adults with an estimated glomerular filtration rate (eGFR) of less than 60 ml/min/1.73m^2
- Heart failure
- Liver failure
- Diabetes mellitus
- Previous history of AKI
- Neurological or cognitive impairment
- Age over 65 years

Source: NICE (2013).

As there are many causes of AKI, it is worth thinking about them as pre-renal, intrinsic and post-renal (Table 2). This should help to identify the cause and also plan management. Pre-renal causes of AKI induce renal ischaemia from reduced blood flow to the kidney. Intrinsic AKI is caused by damage to the kidney itself, either at the glomerulus, nephron or interstitium. Post-renal AKI is caused by urinary tract obstruction, and therefore the kidney is producing urine, but this is unable to be drained from the body. This causes a back-up of urine within the kidney, and hence, reduces its ability to function. Remember there maybe more than one cause of AKI present.

Clinical assessment of AKI

AKI in primary care is most likely to be seen in the acutely unwell patient. When assessing a patient who is acutely unwell and might have AKI, it is important to focus on volume state, sepsis, hypotension and medication review. Assessing a patient's volume state should be done though history taking, observations and clinical examination. There

Table 2. Aetiology of AKI.	
Pre-renal causes: Ischaemia from renal hypotension	
Volume depletion	• *Loss of volume:* Haemorrhage, burns • *Reduced intake:* Dehydration, reduced oral intake • *Increased output:* Vomiting, diarrhoea, over diuresis
Oedematous state	Cardiac failure, liver failure, nephrotic syndrome
Renal hypoperfusion	NSAIDs, ACE inhibitors, ARBs, renal artery stenosis
Vasodilatation	Sepsis, anaphylaxis
Intrinsic causes: Damage to glomerulus, nephron or interstitium	
Glomerular	Glomerulonephritis
Tubular	• *Ischaemia:* Acute tubular necrosis (prolonged hypoperfusion) • *Toxins:* Computed tomography or X-ray contrast, medications such as proton pump inhibitors, lithium and metformin, myoglobin from rhabdomyolysis • *Crystals:* Urate and oxalate • *Metabolic:* Immunoglobulin light chains from myeloma
Interstitial nephritis	• *Drugs:* NSAIDs, some antibiotics • Infection • Lymphoma • *Granulomatous:* tuberculosis, sarcoidosis
Vascular	Vasculitis (usually anti-neutrophil cytoplasmic antibody- related), malignant hypertension, renal artery stenosis
Post renal: Obstruction	
Extra-renal obstruction	• *Bladder outflow obstruction:* Enlarged prostate, blocked catheter, bladder stones, faecal impaction • *Ureteric obstruction:* Pelvic malignancy, retroperitoneal fibrosis, calculi, ureteric strictures
Intra-renal obstruction	Clots, renal tumour, renal stones

Source: Hilton (2006).

is no single clinical test that can determine volume state; it is based upon a range of clinical findings.

History

Particular focus in the history should be on volume state with careful questioning about oral intake or excessive output, such as gastrointestinal losses. Some key questions that can help work out volume state are:

- Can the patient drink/take fluids?
- Is the patient thirsty?
- What is the urine output?

Asking about thirst as a marker of volume state is useful; however, it is less reliable in the elderly. This is due to physiological changes in the thirst control centres with age. Therefore, this question is probably most useful in adults under the age of 70 years.

Some focused questions to elicit the presence of sepsis would also be important. These include asking about fever, rigors and confusion.

Examination

Clinical examination is useful in assessing volume state, but individual clinical findings can be non-specific. However, together they should provide relevant information (Academy of Medical Royal Colleges, 2011):

- Postural vital signs: Most specific for hypovolaemia is supine hypotension (systolic blood pressure of less than 95 mmHg)

- Dry axillae
- Moistness of mucus membranes
- Presence of tongue furrows
- Sunken eyes
- Jugular venous pressure
- Reduced skin turgor: Look for persistent tenting of the skin when gently pulled upwards on the arm or abdomen in dehydration
- Reduced conscious level
- Increased capillary refill time (greater than 5 seconds)
- Evidence of peripheral oedema, pleural effusions, ascites or pulmonary oedema

Other important clinical findings when assessing for causes of AKI include: a palpable bladder; swollen joints; rashes; and any signs of infection. There is a well-recognised link between AKI and sepsis. Sepsis is a life-threatening condition caused by the body's own response to infection by injuring its own tissues (Singer et al., 2016). It is important to identify signs of sepsis by performing blood pressure, pulse rate, respiratory rate, and assessing consciousness level. If sepsis is suspected then you should consider immediate referral to secondary care, if appropriate (NICE, 2016).

Investigations

Blood tests should be performed if you suspect that a patient could have AKI, particularly for those with co-morbidities and who are taking anti-hypertensive medication, metformin and other nephrotoxic medications. The NICE AKI guideline (NICE, 2013) recommends testing serum creatinine in patients with an acute illness and any one of the conditions mentioned in Box 2.

Blood tests with a serum creatinine help to diagnose AKI (Table 1). If the level of serum potassium is greater than 6.5 mmol/L, then you should consider immediate referral to secondary care (see Fig. 1). Specialist blood tests such as antinuclear antibody or myeloma screens should only be done once AKI is confirmed and there is a high clinical index of suspicion that there is an intrinsic cause for AKI. Urine dipstick testing should be performed on all patients with suspected AKI. This will help diagnose urine infection, as well as identifying microscopic blood and protein in the urine.

An urgent ultrasound scan of the renal tract within 24 hours of assessment is advised by NICE (2013) if no other cause for the AKI can be found or the patient is at risk of urinary tract obstruction. In primary care, this will usually require acute admission.

Medication review

During the clinical assessment of a patient with possible AKI, medication review is vital. If a patient is taking a medication that either reduces the blood flow to the kidney through lowering blood pressure or another mechanism, the medication should be temporarily stopped to try and reduce the severity of AKI. Reviewing medications and stopping appropriate ones at time of illness is probably the best way to help prevent AKI.

The key medications that should be reviewed in the acute setting are (Griffith, Ashley, Blackman, et al., 2015; Think Kidneys, 2016a):

- Angiotensin-converting enzyme (ACE) inhibitors or angiotensin receptor blockers (ARBs): Reduce glomerular perfusion and vasodilate efferent arteriole
- Anti-hypertensive medication: Reduce glomerular perfusion
- Non-steroidal anti-inflammatory drugs (NSAIDs): Inhibit the vasodilatation of the afferent arteriole
- Diuretics: Cause electrolyte disturbances
- Metformin: Causes lactic acidosis

Consider medications that might build-up in a patients system if they have worsened renal function, and potentially reach levels which may produce toxic effects such as digoxin, lithium, glycaemic control medications, and opioids. Often there is no need to stop these acutely, but it is important to review them with the patient with first review occurring within 48 hours.

Box 2. Investigating for CKD.

Investigate for AKI by measuring serum creatinine and comparing with baseline, in adults with acute illness if any of the following are likely or present:

- CKD with an eGFR of less than 60 ml/min/1.73m^2– on the CKD register at the practice
- Heart failure
- Liver failure
- Diabetes
- History of previous AKI
- Oliguria (urine output less than 0.5 ml/kg/hour) - but this is hard to determine in primary care
- Neurological or cognitive impairment that limits fluid access due to reliance on a carer
- Hypovolaemia
- Use of medications with nephrotoxic potential NSAIDs, ACE inhibitors, ARBs and diuretics
- Use of iodinated contrast within the last week – ask if the patient has had an X-ray or computed tomography scan with an injection given at the same time
- Symptoms or history of urological obstruction, or conditions which might lead to obstruction
- Deteriorating early warning scores
- Aged 65 years old or over

NICE (2013) CG169 Acute Kidney Injury: Prevention, detection and management. London: NICE. Available from www.nice.org.uk/guidance/cg169. Reproduced with permission. The information was correct at the date of publication.

AKI warning stage test result Confirm **or** refute automated AKI Test Result by comparing patient's current creatinine **within clinical context** against baseline creatinine	**Clinical context within which blood test taken#** If clinical context is unknown, then assume high pre-test probability until proven otherwise	
	LOW pre-test probability of AKI Stable clinical context	**HIGH pre-test probability of AKI** Context of acute illness
AKI warning Stage 1 Current creatinine greater than 1.5 x baseline level (*or* creatinine rise greater than 26 micromol/L in less than 48 hrs)	**Consider clinical review within 72 hours of e-alert*** **If AKI confirmed**→ manage as per Box 3	**Consider clinical review within 24 hours of e-alert*** **Likely Stage 1 AKI**→ manage as per Table 3
AKI warning Stage 2 Current creatinine greater than2 x baseline level	**Consider clinical review within 24 hours of e-alert*** **If AKI confirmed**→ manage as per Table 3	**Consider clinical review within 6 hours of e-alert*** **Likely Stage 2 AKI**→ manage as per Table 3
AKI warning Stage 3 Current creatinine greater than3 x baseline level (*or* creatinine 1.5 x baseline and greater than 354 micromol/L)	**Consider clinical review less than 6 hours of e-alert*** **If AKI confirmed**→ consider admission	**Consider immediate admission*** Likely Stage 3 AKI

#Clinical context
Why was the blood test taken?
- Routine chronic disease monitoring
- Drug monitoring
- Assessment of acute illness

Creatinine rise within stable clinical context **may** reflect unstable CKD instead of AKI, especially if **longer time period** between current and baseline creatinine.

***AKI Risk Factors/Clinical Features Prompting Earlier Review**
- Poor oral intake/urine output
- Evidence of hyperkalaemia, especially if moderate (serum potassium 6.0-6.4 mmol/l) or severe (serum potassium of 6.5 mmol/l or greater)[¥]
- Known history of CKD stages 4 and 5 or history of kidney transplant
- Deficient immunity
- Frail with co-morbidities (CKD, diabetes, heart failure, liver disease, neurological or cognitive impairment)
- Past history of AKI
- Suspected intrinsic kidney disease
- Suspected urinary tract obstruction

Figure 1. Acute kidney injury: Recommended response times to AKI warning stage test results for adults in primary care. *(Source: Griffith et al., 2015).*

Dreischulte, Morales, Bell, and Guthrie (2015) showed the use of NSIADs, ARBs or ACE inhibitors and diuretics (the 'triple whammy') increases the risk of AKI in those over the age of 75 years and with pre-existing renal impairment. This study showed that AKI occurred more often in triple therapy compared with dual therapy (NSAIDs with either a diuretic or renin-angiotensin aldosterone antagonist). Therefore, it is not advisable to have patients on this combination without regular review or follow-up and renal function monitoring.

Warning stage test results

In June 2014, NHS England issued a Patient Safety Alert to highlight that there needed to be standardisation of the definition of AKI among pathology services within England. This has led to alerts being added to patient's blood test results to highlight the possible presence of AKI and prompt earlier detection and management (Think Kidneys, 2016b). Within secondary care these alerts have already been activated. They are now beginning to be activated for patients who have had blood tests done in the community. These alerts highlight the presence of AKI and give a stage, which is consistent with the KDIGO definition (Table 1). On average a GP practice will get about one alert per month for every 1500 patients on their list (Think Kidney, 2016b). These alerts need to be reviewed within the clinical context (Figure 1) as often this is the first blood test for a prolonged period of time.

Some patients may not have had a blood test for creatinine for a long time, and so trying to interpret whether this is a true or false alert is important. If the patient is acutely unwell, then AKI is more likely. If there is a greater than 50% creatinine rise from the previous one, then it is also likely to be true AKI. In a patient without any previous estimation of renal function, clinical judgement is required and if you are concerned about AKI, repeat the patient's renal function test to try to identify a trend in renal function.

Some important causes of false alerts are as follows: (Think Kidneys, 2016b):
- Progression of CKD
- Trimethoprim: This increases serum creatinine without changing underlying kidney function
- Pregnancy: Creatinine naturally falls during pregnancy; after delivery the creatinine will return to normal

Management

Management of AKI is usually supportive offering fluid replacement, treatment of infection or stopping appropriate medications. Fluid replacement should be encouraged if the patient is hypovolaemic. The patient has to be well enough to take fluid orally. Medications should be reviewed, especially ones started within the preceding week. If infection is present, appropriate treatment should be commenced. For those patients who remain in the community, repeat blood tests in 24–48 hours to check renal function. Table 3 summarises recognition and management of AKI in primary care.

Table 3. Recognising and responding to acute kidney injury in primary care.[a]

'Think' Cause	'Think' Medication[b]	'Think' Fluids	'Think' Review[c]
History of acute Illness? • Think sepsis • Think hypotension Intrinsic kidney disease? (e.g. vasculitis) • Think urinalysis Urinary tract obstruction?	Any medication which could **exacerbate** AKI? Consider withholding: • NSAIDs • Diuretics • Anti-hypertensive medication Any medication that may **accumulate** and cause harm during AKI? Any **new** medication that may **cause** AKI? (e.g. drug-induced tubulo-interstitial nephritis)	What is the patient's volume status? If hypovolemia present: • When did patient last pass urine? • Can the patient increase fluid intake? • Is admission for intravenous fluid replacement and monitoring required? Does the patient have and/or need carer support?	Does the patient need acute admission? If not, when will you review? Have you ensured handover?[c]

[a]Refer to main guidance document: www.thinkkidneys.nhs.uk/aki/wp-content/uploads/sites/2/2016/03/Responding-to-AKI-Warning-Stage-Test-Results-for-Adults-in-Primary-Care.pdf
[b]Refer to medicines optimisation toolkit for primary care www.thinkkidneys.nhs.uk/aki/medicines-optimisation-for-aki
[c]Refer to overarching principles in communication of diagnostic test results https://www.england.nhs.uk/patientsafety/discharge
Reproduced from Think Kidneys (2016b). Acute kidney injury. Best practice guidance: Responding to AKI warning stage test results for adults in primary care, with permission from the UK Renal Registry.

Follow-up

Follow-up in primary care of patients with AKI is important, due to the potential for existing CKD to worsen or for the patient to develop new CKD. NICE advises follow up for 2 to 3 years following all stages of AKI, even if the creatinine returns to baseline. Think Kidneys (2016b) advises that, as a minimum, serum creatinine and urine albumin-to-creatinine ratio should be performed 3–6 months following an episode of AKI.

If a patient is admitted with AKI then Box 3 shows the ideal information that should be included in the discharge summary from secondary care to guide follow-up within primary care (Think Kidneys, 2016b). Ideally AKI should be coded as a maker of vulnerability and a practice should keep an AKI register.

Restarting medications after AKI

When considering the reintroduction of temporarily suspended medications, the initial indication for the medication should be reviewed. There should also be a discussion with the patient or carers about the pros and cons of restarting medication. If there was a specific contra-indication reported to one of the stopped medications, then an alternative should be considered, for example, severe bilateral renal artery stenosis precluding the on-going use of ACE inhibitors or ARBs (Think Kidneys, 2016b). For patients previously on ACE inhibitors or ARBs with CKD and albuminuria, these should be recommenced unless the level of serum potassium is greater than 5 mmol/L (NICE, 2014).

Box 3. Gold standard discharge summary information following an episode of AKI

Cause(s) of AKI
- Was the AKI present on admission, or was it hospital acquired?
- Highest AKI stage (stage 1, 2 or 3)
- Intensive care admission: yes or no
- Was renal replacement therapy required: yes or no
- Were any drugs stopped during admission (e.g. ACE inhibitors, ARB, diuretics, other blood pressure lowering drugs)? If so: restarted prior to discharge yes/no

Information on recovery and follow-up
- Serum creatinine at discharge. Stable or still improving at time of discharge?
- Requirement for follow-up including repeat laboratory test and who is responsible for the tests – this should include frequency, type of test and for how long
- Specific instructions on medicines management especially those medications that can be resumed and those to be avoided long term.
- Discharge summaries should offer additional sources of information to primary care on AKI. This may include electronic links or written information.

Reproduced from Think Kidneys (2016). Discharge summaries for patients whose hospital admission included an episode of AKI: minimum data content, with permission from the UK Renal Registry.

For patients with heart failure, medications, on which they were previously stable, should be slowly re-introduced. They can be titrated as blood pressure and fluid balance allow, as long as there are no contra-indications. If the patient is known to the heart failure team, then their support with titration of medication can be very valuable (Think Kidneys, 2016d).

Patients with essential hypertension, and who have had an episode of AKI and medications withheld, should be reviewed within 6 weeks of the episode of AKI. Blood pressure should be checked with either ambulatory or home monitoring to guide anti-hypertensive medication as needed (Think Kidneys, 2016). It is important to follow the NICE/British Hypertension Society guidance for anti-hypertensive medication, especially for patients on single therapy. For example, patients over the age of 55 years and black people of African or Afro-Caribbean origin should be offered a calcium channel blocker as first-line treatment (NICE and British Hypertension Society, 2011). If ACE inhibitors or ARBs are to be commenced, then serum potassium should have a level less than 5 mmol/L and creatinine should be checked at 1 to 2 weeks.

If a patient with previously treated essential hypertension has a clinic blood pressure of 140/90 mmHg or home blood pressure of 130/85 mmHg, regular follow-up should be offered for 12 months, as it can on occasions take some time for blood pressure to return to previous levels after recovery from an acute illness (Think Kidneys, 2016d).

AKI and CKD

Patients who have suffered an episode of AKI should be followed up in the long term, as having an episode of AKI can increase the risk of developing CKD and the associated increased cardiovascular risk that accompanies that diagnosis. It is important not to think of AKI and CKD as two separate conditions, but rather as interconnected syndromes. AKI can lead to CKD, CKD can increase susceptibility to AKI and both AKI and CKD can lead to increased cardiovascular risk factors (Chawla, Eggers, Star, and Kimmel, 2014). Even in minor AKI there is an increased risk of developing CKD, highlighting the importance of follow-up in primary care (Wannacott et al., 2014).

If new onset CKD does occur, then management of this should be guided by the NICE guideline for CKD (NICE, 2014). If there is evidence of significant renal function decline, consider referral to nephrology.

Prevention of AKI

The majority (65%) of AKI occurs in the community and it is recognised that some of this AKI is preventable. It is important to recognise at-risk patient groups and manage them accordingly with prevention of hypovolaemia, and careful drug selection and monitoring (NICE, 2013).

Patient education can be challenging. An Ipsos MORI study in 2014 showed that only one in two of the general population knew that their kidneys made urine (Slevin and Taylor, 2015). Therefore, tailoring education about renal health and medications to each patient individually is important. Think Kidneys has some useful patient education leaflets on their website.

Sick day guidance

Medicine sick day guidance is based around individual patient education about their medications. At a time of illness such as diarrhoea, vomiting or fever, patients can adjust their own medications to reduce potential harm from certain medication groups (Box 4).

Box 4. Sick day guidance on stopping other medications.

1. NSAIDs: These inhibit the vasodilatation of the afferent arteriole and can increase the risk of AKI
2. Drugs that lower blood pressure and might increase the risk of AKI through reducing glomerular perfusion. This includes ACE inhibitors and ARBs (which also vasodilate efferent arterioles), diuretics (which also cause electrolyte disturbances) and other anti-hypertensive medication
3. Drugs that might accumulate due to reduced kidney function, and therefore, increase adverse effects, such as metformin (lactic acidosis), sulfonylureas (hypoglycaemia) and trimethoprim (hyperkalaemia)

Source: Griffith et al. (2015).

Many GP surgeries now stock sick day guidance cards that can be given to patients at high risk of AKI, but it is important to select the right patients to use these cards and educate them appropriately to minimise harm.

There does need to be some caution with the sick day guidance, especially in patients with moderate-to-severe heart failure. There are also concerns that some patients may stop taking some medications, as they think it will harm their kidneys in the absence of acute illness. Furthermore, patients may not restart medications that are beneficial after they have been stopped for sick days. It can also be difficult to stop drugs for patients who have pre-filled medication dispensers such as Nomad trays or Dosette boxes. Therefore, guidance on sick day rules should be based on an individual risk assessment.

Key points

- AKI is common, harmful, costly and preventable
- Only one-in-two people know their kidneys make urine
- Sixty-five percent of AKI occurs in the community
- AKI should be considered in a patient who is acutely unwell with co-morbidities such as CKD, diabetes and heart failure
- Pre-renal causes are responsible for 70% of AKI
- Primary care follow up of AKI (even with creatinine returning to baseline) should be for 2 to 3 years to monitor for development or worsening of CKD

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