



Acute care toolkit 12

Acute kidney injury and intravenous fluid therapy September 2015

Acute kidney injury (AKI) is a global health issue that is associated with increased morbidity and mortality, prolonged hospital stays and spiralling costs.¹ The majority of causes of AKI are secondary to sepsis and hypotension, and are potentially reversible if the patient recovers.

However, it is now recognised that patients who survive AKI are at risk of chronic kidney disease (CKD) and the subsequent increased risk of cardiovascular disease.² Prevention and prompt treatment of AKI are therefore essential to reduce patient risk and preserve kidney function. AKI has been proposed as a quality metric of patient care. This toolkit reflects clinical best practice in the management of patients with, or at risk of, AKI and includes guidance on intravenous (IV) fluid therapy.

Definition and staging of AKI

New definitions have been developed to reflect the fact that even seemingly minor changes in serum creatinine (SCr) levels are associated with a significant increase in risk of mortality. The term 'acute kidney injury' replaces 'acute renal failure' and is defined by criteria that are dependent upon either rises in SCr level or reductions in urine output.³ AKI should be regarded as a spectrum of injury (Table 1) that may progress to organ failure and the need for renal replacement therapy, with associated increased mortality.⁴

Table 1 Definition and staging of AKI³

AKI stage	SCr criteria*	Urine output criteria
1	SCr increase $\geq 26 \mu\text{mol/L}$ within 48 h or SCr increase $\geq 1.5\text{--}1.9$ fold from baseline	$<0.5 \text{ mL/kg/h}$ for 6 h (consecutive)
2	SCr increase $\geq 2\text{--}2.9$ fold from baseline	$<0.5 \text{ mL/kg/h}$ for 12 h
3	SCr increase ≥ 3 fold from baseline or SCr increase $\geq 354 \mu\text{mol/L}$ or initiated on RRT (irrespective of AKI stage at time of initiation)	$<0.3 \text{ mL/kg/h}$ for 24 h or anuria for 12 h

RRT = renal replacement therapy; SCr = serum creatinine

*The baseline SCr level is defined as the lowest within 7 days or, if this result is not available, the lowest within the previous 3 months

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Causes of AKI

AKI has many causes (Fig 1), some of which will require immediate referral to nephrology, urology or radiology teams. It is therefore essential that the cause of AKI is identified in all patients; otherwise, treatment may be delayed, resulting in the addition of further injury to the original insult.

The causes of AKI when it occurs in hospital are similar in both developing and developed countries. However, the causes of AKI in the community differ in developing countries from developed countries; the former include pre-eclampsia, haemolytic uraemic syndrome, malaria, leptospirosis, snake bites, and dehydrating illnesses including dysentery.

AKI in the setting of cardiac failure may be especially difficult to treat. Overall, the aim will be to restore the best possible perfusion to both heart and kidneys. In the setting of obvious fluid overload, diuretic therapy may offload the heart adequately to improve both cardiac output and renal perfusion. In the hypotensive patient with poor cardiac output, therapeutic options may be limited. Some studies have demonstrated temporary benefit from haemofiltration; however, in this situation senior input is required and a review of the overall patient benefit should be initiated. This should include discussion with the patient and carer(s) about appropriate levels of care.

Risk factors for AKI

AKI occurs most commonly in at-risk patients, who either are acutely ill or have had major surgery. Recognition of those at risk of AKI is important, because patients may develop AKI while in hospital or in the community⁵ unless specific preventative actions are taken. These may be as simple as ensuring that adequate hydration is maintained, especially in vulnerable patients who are unable, for whatever reason, to actively sustain their own hydration. A number of risk factors for AKI have recently been identified (Box 1).⁶

Box 1 Risk factors for AKI

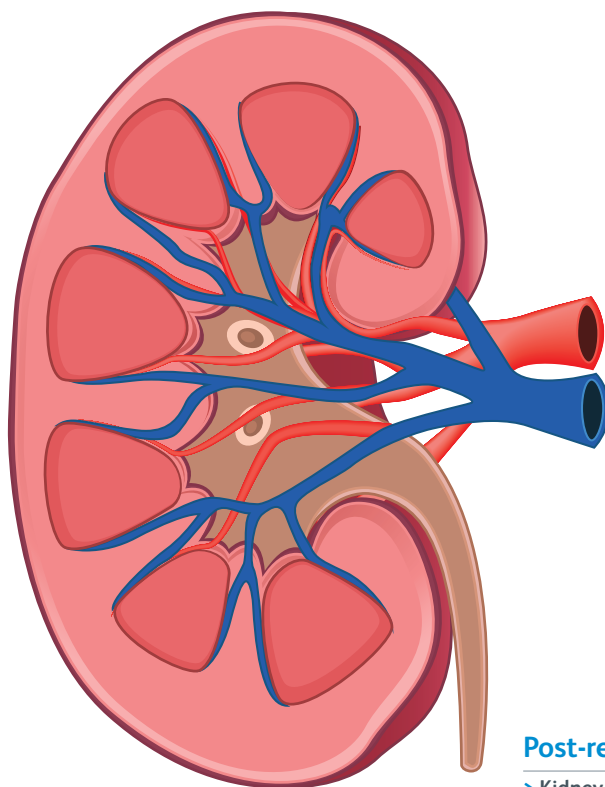
- > Age >75 years
- > Pre-existing CKD (eGFR <60 mL/kg/1.73 m²)
- > Previous episode of AKI
- > Debility and dementia
- > Heart failure
- > Liver disease
- > Diabetes mellitus
- > Hypotension (mean arterial pressure <65 mmHg, systolic pressure <90 mmHg)
- > Sepsis
- > Hypovolaemia
- > Nephrotoxins, eg gentamicin, NSAIDs, iodinated contrast
- > Antihypertensives in setting of hypotension, eg ACE inhibitors, loop diuretics

ACE = angiotensin-converting enzyme; eGFR = estimated glomerular filtration rate; NSAIDs = non-steroidal anti-inflammatory drugs

Fig 1 Causes of AKI

Intrinsic AKI

- > Acute tubular injury
 - ~ Prolonged pre-renal AKI
 - ~ Rhabdomyolysis
 - ~ Haemoglobinuria
 - ~ Nephrotoxins
 - Iodinated contrast
 - NSAIDs
 - Gentamicin
- > Tubulointerstitial injury
- > Glomerulonephritis
- > Myeloma
- > Vasculitis
 - ~ Lupus
 - ~ ANCA associated



Pre-renal AKI

- > Sepsis
- > Hypovolaemia
 - ~ Haemorrhage
 - ~ Burns
 - ~ Vomiting/diarrhoea
 - ~ Diuretics
- > Hepatorenal syndrome
- > Cardiac failure
- > Hypotension
 - ~ Medications

Post-renal AKI

- > Kidney stones
- > Prostatic hypertrophy
- > Tumours
- > Retroperitoneal fibrosis

ANCA = antineutrophil cytoplasmic antibody;
NSAIDs = non-steroidal anti-inflammatory drugs

Work is ongoing to develop and validate AKI risk calculators for both primary and secondary care. In the interim, existing risk tools are important to raise awareness of AKI and promote its earlier detection.

- > As AKI occurs in acutely ill patients, a National Early Warning Score (NEWS) of 5 or greater should trigger the measurement of fluid balance, including urine output, and checking of kidney function.⁷
- > The Sepsis Six care bundle promotes aggressive management of sepsis within the first hour and includes hourly measurement of urine output, which should prompt the measurement of kidney function.⁸
- > Conversely, patients developing AKI should be monitored closely for worsening physiological parameters and signs of sepsis.

These risk tools should be used in the community, allowing earlier identification of patients at risk of deteriorating. In the community, care plans are being implemented for the 2% most vulnerable patients, and measures around preventing AKI should be incorporated. An example of this is developing care plans for patients at risk of AKI, such as those with ileostomies. The advice would focus on maintaining good fluid intake if a patient developed high output from the stoma, and monitoring urine output.

Clinical presentation

Patients with most forms of AKI lack specific symptoms and signs early in the course of the injury, and detection requires a high index of suspicion in those at risk. The NCEPOD report demonstrated that the diagnosis of AKI was delayed in 43% of patients.⁹ Introduction of the AKI warning (see below) and the development of electronic alerts (e-alerts) for AKI should promote earlier recognition of AKI and hopefully improve the quality of patient care.

In March 2015, all NHS trusts in England mandated that biochemistry department laboratory information systems must issue an AKI warning, based on rises in SCr level, for all patients in secondary care with AKI.¹⁰ Individual NHS trusts must determine how the warning is communicated to clinical teams caring for patients, in the form of e-alerts. Some NHS trusts have chosen to develop AKI care bundles that are linked to an interruptive alert on the results server, whereby the clinician must engage with the e-alert. Some trusts send automated alerts by text to clinicians, while others have established AKI outreach teams to respond. The implementation of such e-alerts must be supported with an education package to ensure that an appropriate response occurs. The UK national campaign 'Think Kidneys' provides more information about developing awareness of AKI, including an AKI warning algorithm good practice guide.¹¹

Patients themselves, especially those in high-risk groups (Box 1), should be made aware of the possibility of AKI developing in certain situations. These include when dehydration becomes a real possibility, as is likely to occur with diarrhoeal and vomiting illnesses. Temporary cessation of drugs that may exacerbate hypovolaemia and/or hypotension, eg diuretics or angiotensin-converting enzyme (ACE) inhibitors, should be encouraged. It is, however, critical that the need for these medications is reviewed and they should be reintroduced when the acute illness is resolving, especially when they are being used in the treatment of cardiac failure. In patients at risk of AKI, the use of iodinated contrast media should be considered carefully, and guidelines consulted regarding safe administration of the contrast.¹²

It is important to maintain a high index of suspicion in patients without an obvious precipitating event for the rarer forms of AKI. This includes conditions such as vasculitis and tubulointerstitial nephritis, which may occur in association with systemic symptoms such as rash, fever, lethargy and joint pain.

Differentiation of CKD and AKI

Awareness of previous biochemical results will make the differentiation simple. Pre-existing CKD is a risk factor for AKI, and deteriorating biochemistry against a background of known abnormal renal function clinches the diagnosis. When previous blood test results are not available, the clinical presentation may well indicate AKI, eg hypovolaemia and sepsis. Other biochemical features, such as coexistent anaemia, hyperphosphataemia and hypocalcaemia, may suggest CKD. An ultrasound scan showing small kidneys suggests CKD, whereas normal-sized, echo-'bright' kidneys are more suggestive of a form of AKI.

Investigation of AKI

Initial investigations include:

- > urea and electrolytes, and creatinine
- > bicarbonate
- > full blood count – if platelets low, request blood film / lactate dehydrogenase (to diagnose haemolytic uraemic syndrome / thrombotic thrombocytopenic purpura)
- > liver function tests (to diagnose hepatorenal syndrome)
- > $\text{Ca}^{2+}/\text{PO}_4^{3-}$ (to diagnose myeloma)
- > creatine kinase (to diagnose rhabdomyolysis)
- > blood cultures if sepsis suspected
- > urinalysis – if blood, protein, leucocytes or nitrites, send midstream urine
- > ultrasound scan of renal tract and bladder:
 - if obstruction suspected, scan within 24 h
 - if pyonephrosis suspected, scan within <6 h.

Management of AKI

The mainstay of managing AKI is supportive therapy with close monitoring. A simple acronym to assist with immediate management is STOP AKI (Table 2, page 4), which focuses on treating sepsis aggressively if present, restoring haemodynamic stability, avoiding nephrotoxins and preventing iatrogenic harm to the patient, and identifying the underlying cause. Further treatment will be required to manage the complications of AKI (Table 3, page 4) and to relieve renal tract obstruction if present.

Patients with most forms of AKI lack specific symptoms and signs early in the course of the injury, and detection requires a high index of suspicion in those at risk.

Table 2 STOP AKI management acronym IV fluid therapy

STOP AKI	Response
Sepsis	<ul style="list-style-type: none"> > Sepsis Six care bundle: <ul style="list-style-type: none"> - blood cultures - urine output – hourly (urea and electrolytes) - fluids – IV - antibiotics – IV - lactate and haemoglobin - oxygen – high flow > Identify and treat source of sepsis
Toxins	<ul style="list-style-type: none"> > Stop/avoid potential nephrotoxins: <ul style="list-style-type: none"> - gentamicin - NSAIDs - iodinated contrast
Optimise BP	<ul style="list-style-type: none"> > Volume status assessment: <ul style="list-style-type: none"> - IV fluids > Hold BP-lowering medication > Consider vasopressors
Prevent harm	<ul style="list-style-type: none"> > Treat complications > Identify the cause and investigate > Review: <ul style="list-style-type: none"> - all medications - fluid management plan

BP = blood pressure; IV = intravenous;
NSAIDs = non-steroidal anti-inflammatory drugs

Any dehydrated patient is at risk of AKI, particularly in the setting of acute illness. Accurate measurement of fluid balance to include daily weights (if practical) is essential. All patients must be encouraged and, if necessary, helped to maintain their oral fluid intake. If oral replacement is not possible, other mechanisms of fluid replacement must be sought. For patients who are fasting for any protracted period beyond 12 h or in whom losses of fluid cannot be matched by oral intake, parenteral fluids must be commenced. Consider whether the patient may face haemodialysis in the future, and avoid veins in the non-dominant arm that may be used to create an arteriovenous fistula. Prior to any prescription of IV fluids, the patient should have a volume assessment that includes the following parameters:

- > capillary refill
- > pulse rate
- > blood pressure – check for postural drop
- > jugular venous pressure
- > respiratory rate
- > oedema – pulmonary or peripheral
- > passive leg raising test to assess volume responsiveness
- > fluid balance chart review
- > weight (trend important).

Passive leg raising is most commonly practised on critical care units, where it can be used to promote venous return and the physiological response in blood pressure can be recorded. A rise in blood pressure indicates that the patient is hypovolaemic and that further fluid resuscitation is required. It is important to remember that certain drugs affect normal physiological responses: for instance, beta blockers affect the pulse response, and postural hypotension may occur as a complication of a number of medications, including many antihypertensives.

Table 3 Complications of AKI

Complication of AKI	Management
Hyperkalaemia (mild): K ⁺ 5.5–5.9 mmol/L	<ul style="list-style-type: none"> > Consider cause and treatment if indicated <i>Underlying cause must be identified and treated, because therapy results only in temporary intracellular shift of potassium¹³</i>
Hyperkalaemia (moderate): K ⁺ 6.0–6.4 mmol/L	<ul style="list-style-type: none"> > Treatment guided by clinical scenario, ECG and rate of rise of K⁺ level > Glucose (25 g) over 15 min: <ul style="list-style-type: none"> 50 mL 50% glucose or 125 mL 20% glucose with 10 units soluble insulin (lasts 4–6 h) > Salbutamol 10–20 mg nebulised (10 mg if history of ischaemic heart disease) Avoid if tachyarrhythmias present
Hyperkalaemia (severe): K ⁺ ≥6.5 mmol/L Monitor ECG in high-dependency area if acute ECG changes present	<ul style="list-style-type: none"> > IV calcium 6.8 mmol: <ul style="list-style-type: none"> 10 mL 10% calcium chloride or 30 mL 10% calcium gluconate Large vein over 5–10 min <i>Seek expert advice from renal or ICU team if ECG fails to normalise after one dose</i>
Acidaemia: pH <7.25	<ul style="list-style-type: none"> > IV sodium bicarbonate can be considered, but only with expert supervision > Consideration of critical care/ICU referral
Pulmonary oedema	<ul style="list-style-type: none"> > Sit patient up > O₂ 15 L/min via reservoir mask > IV GTN (50 mg in 50 mL 0.9% sodium chloride): commence with 2 mL/h and titrate up to 20 mL/h maintaining systolic BP >95 mmHg > Furosemide only if patient is haemodynamically stable and well filled intravascularly
Uraemic encephalopathy / pericarditis	<ul style="list-style-type: none"> > Renal replacement therapy

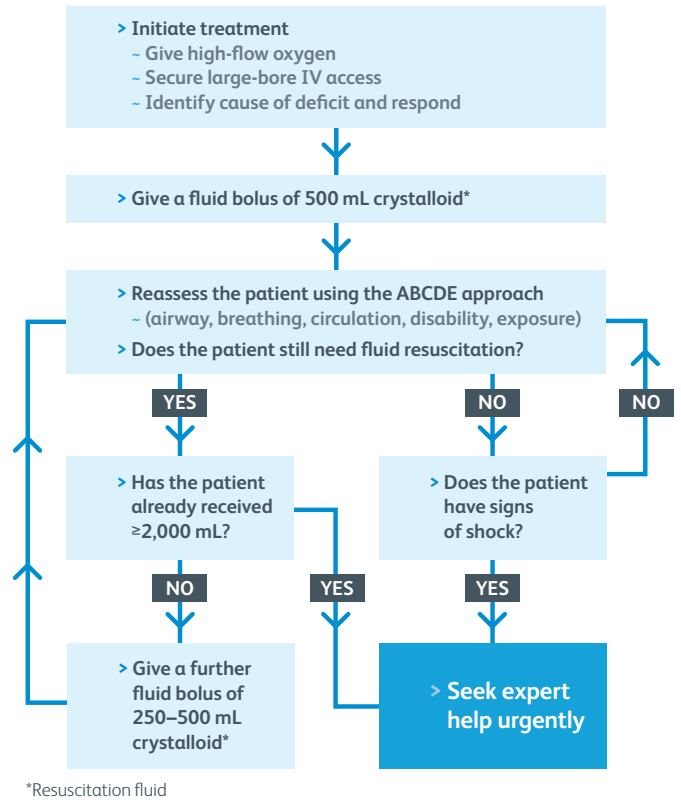
BP = blood pressure; ECG = electrocardiogram; GTN = glyceryl trinitrate; ICU = intensive care unit; IV = intravenous

The National Institute for Health and Care Excellence (NICE) recommends that assessment of a patient’s fluid balance should be part of every ward round.¹⁴ It also recommends that IV fluid therapy is classified as resuscitation, replacement or routine maintenance (Table 4), and that any prescription should identify clearly which type of IV fluid therapy the patient is receiving.

Table 4 Classification of IV fluid therapy

Type of fluid therapy	Definition	Example of fluid
Resuscitation	Re-establishes haemodynamic stability through restoring intravascular volume	<ul style="list-style-type: none"> > Balanced crystalloid <ul style="list-style-type: none"> ~ Hartmann’s solution ~ Ringer’s acetate ~ Plasma-Lyte 148 > Crystalloid <ul style="list-style-type: none"> ~ 0.9% sodium chloride
Replacement	Provides daily maintenance requirements and replacement of any ongoing abnormal losses	<ul style="list-style-type: none"> > Balanced crystalloid <ul style="list-style-type: none"> ~ Hartmann’s solution ~ Ringer’s acetate ~ Plasma-Lyte 148 > Crystalloid <ul style="list-style-type: none"> ~ 0.9% sodium chloride
Routine maintenance	Provides daily maintenance requirements	<ul style="list-style-type: none"> > 0.18% sodium chloride / 4% dextrose > 0.45% sodium chloride > 5% dextrose

Fig 2 Algorithm for initial fluid resuscitation



NICE recommends that fluid resuscitation is best approached with a crystalloid solution rather than a colloid, and has produced an algorithm to guide the initial administration (Fig 2). Most experts would recommend a balanced crystalloid (eg Hartmann’s, lactated Ringer’s or Plasma-Lyte 148), because excessive amounts of 0.9% sodium chloride can result in hyperchloraemic metabolic acidosis. The only exception is rhabdomyolysis, when 0.9% sodium chloride would be preferred initially because of the risk of hyperkalaemia. It is important to know the electrolyte content of the fluid that is prescribed (Table 5).

To facilitate the fluid challenge, an IV cannula of adequate size to infuse the required fluid at a high enough rate must be used. This usually means at least a pink cannula and administration of 250–500 mL fluid over 5–15 minutes. It is important to develop guidelines for IV access, because delays can occur in the administration of fluids (and antibiotics) owing to poor access or lost access.

Table 5 Composition of fluids

Type of fluid	Na ⁺ mmol/L	K ⁺ mmol/L	Cl ⁻ mmol/L	Ca ²⁺ mmol/L	HCO ₃ ⁻ precursor mmol/L	Glucose mmol/L	Osmolarity mOsm/L
Resuscitation fluids							
Hartmann’s	131	5	111	2	29 (lactate)	0	278
Lactated Ringer’s	130	4	109	1.4	28 (lactate)	0	273
Plasma-Lyte 148	140	5	98	0	27 (acetate) 23 (gluconate)	0	295
0.9% sodium chloride	154	0	154	0	0	0	308
Replacement or routine maintenance fluids							
0.45% sodium chloride	77	0	77	0	0	0	154
0.18% sodium chloride / 4% dextrose	31	0	31	0	0	222.2	284
5% dextrose	0	0	0	0	0	277.8	278

Responsiveness to the fluid challenge should be assessed by regular monitoring of blood pressure and pulse rate.

- > If there is an improvement in either or both that is poorly sustained, the fluid challenge should be repeated.
- > If haemodynamic stability is restored and the patient is deemed euvoalaemic, the type of IV fluid prescribed should be reviewed.
- > If there is no improvement after two accurately performed fluid challenges, the patient should be reviewed and, when appropriate, their care should be escalated for senior review and possible critical care involvement for vasopressor support.

NICE has indicated the salts and fluid volume necessary for routine maintenance IV fluid therapy. To avoid ketosis, a source of calorie intake must be available even if, in the short term, this consists of the addition of 5% dextrose to the IV administration of fluid.

It is beyond the remit of this toolkit to review parenteral nutrition or more complex fluid replacement that may be required in specific patient groups. Even for seemingly simple fluid replacement, however, optimal management requires an awareness of the patient's serum biochemistry. It is not satisfactory simply to copy the previous day's prescription of IV fluid. The replete adult patient at stable volume requires 1 mmol/kg of both sodium and potassium, 25–30 mL/kg/day of water and enough calories to prevent ketosis. The type of fluid prescribed can be selected from those listed in Table 5 and will be dependent upon each patient's individual calculated sodium and potassium requirements. Crystalloid solutions can be selected, with pre-added potassium ranging from 20 to 40 mmol/L. In patients receiving IV fluids, electrolytes should initially be checked on a daily basis, and appropriately monitored thereafter.

Table 6 Recommended volume of fluid and rate of administration for routine maintenance IV fluid therapy

Weight (kg)	Fluid requirement (mL/day)	Rate (mL/h)
35–44	1,200	50
45–54	1,500	65
55–64	1,800	75
65–74	2,100	85
≥75	2,400	100 (max)

Rules for prescribing routine maintenance IV fluids include:

- > IV fluid should be given via volumetric pump if a patient is on fluids for over 6 h or if the fluid contains potassium.
- > always prescribe as mL/h, not 'x hourly' bags
- > never give maintenance fluids at more than 100 mL/h
- > never prescribe fluids for more than 24 h at a time.

Referral

The majority of patients with AKI do not require referral to a nephrologist for further care. If a trust does not have an on-site nephrology service, it should establish appropriate patient pathways for referral and transfer. The NICE CG169 AKI clinical practice guideline recommends that the management of AKI should be discussed with a nephrologist as soon as possible, and within 24 h of detection when one or more of the following is present:¹⁵

- > a possible diagnosis that may necessitate specialist treatment (eg vasculitis, glomerulonephritis, tubulointerstitial nephritis or myeloma)
- > AKI with no clear cause
- > inadequate response to treatment
- > complications associated with AKI
- > AKI stage 3
- > a renal transplant
- > CKD stage 4 or 5.

Patient recovery

Patients recovering from a significant episode of AKI may develop profound diuresis, resulting in a free water deficit, hypernatraemia and/or hypokalaemia. During this phase, maintaining patient hydration and replacing electrolytes are of paramount importance. This may require fluid replacement in very large volumes. Accurate fluid balance with daily weights is very important to prevent patients from becoming dehydrated as they recover from AKI.

Discharge planning

Patients who have had an episode of AKI are at risk of CKD in the long term; this risk depends upon the severity of the episode of AKI. Patients' kidney function should be checked prior to discharge. Refer patients to nephrology if they are discharged with an estimated glomerular filtration rate <30 mL/min/1.73 m². Medications should be reviewed prior to discharge, with a plan to reintroduce medications that may have been held during the acute illness, eg antihypertensives or diuretics, at an appropriate time. This may require an early follow-up with the GP. Patients should be told the reason that they developed AKI, and their risk factors. The patient should be an active partner in such discussions.

It is recommended that the GP discharge letter should include severity of AKI, cause of AKI, risk factors for AKI, kidney function on discharge, and advice on whether medications need to be reviewed or reintroduced. This information must be available to the GP at the time of discharge to ensure that patient care is not compromised.

There is no need to monitor renal function at discharge unless potential nephrotoxins are to be reintroduced, but follow-up should be initiated for all patients who have experienced significant AKI.

Education

AKI occurs frequently in the hospital setting and all trainees should acquaint themselves with the common presentations and management pathways early in their career. Critical, life-threatening complications such as hyperkalaemia can be successfully treated by knowing and adhering to the basic protocols. Prevention is far better than cure, and there have already been successful campaigns to remind healthcare workers about the need to ensure that patients are adequately hydrated. In many hospitals, an AKI champion has been identified to help with education and with reinforcing these vital messages.

A number of helpful resources are available to improve education and care for patients with AKI. These include:

- > an app developed by the Royal College of Physicians of Edinburgh that is free to download¹⁶
- > an AKI core competency framework for healthcare professionals, endorsed by the Academy of Medical Royal Colleges¹⁷
- > the RRAPID app and e-book, which contain an AKI risk calculator and a NEWS calculator.¹⁸

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Acute care toolkits

A series of resources to help improve the delivery of acute care. The toolkits look at current problems and suggest a range of recommendations for improving quality.

Coming soon

Acute care toolkit 13: acute care for young adults and adolescents

Acute care toolkit 14: end-of-life care in the acute care setting

Acute care toolkit 12: acute kidney injury and intravenous fluid therapy

Acute care toolkit 11: using data to improve care

Acute care toolkit 10: ambulatory emergency care

Acute care toolkit 9: sepsis

Acute care toolkit 8: the medical registrar on call: Maximising clinical experience, training and patient care

Acute care toolkit 7: acute oncology on the acute medical unit

Acute care toolkit 6: the medical patient at risk

Acute care toolkit 5: teaching on the acute medical unit

Acute care toolkit 4: delivering a 12-hour, 7-day consultant presence on the acute medical unit

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