



ACUTE KIDNEY INJURY

Mobile Application Content

Royal
College of
Physicians
of Edinburgh



Kidney Care

ACUTE KIDNEY INJURY

Recognition

Risk Factors

Risk of AKI = Baseline risk x acute illness risk

Baseline risk may be increased by a history of:

- Chronic Kidney Disease - eGFR <60 ml/min/1.73 m² and/or history of proteinuria
- Age >75 years
- Heart failure
- Liver disease
- Cardiovascular disease (previous MI, stroke, PVD)
- Diabetes mellitus
- Recent use of nephrotoxins, e.g. non-steroidal anti-inflammatory drugs, angiotensin converting enzyme inhibitors, angiotensin receptor blockers, gentamicin, iodinated contrast

Acute illness risk may be increased by:

- Hypotension
(Consider relative hypotension in people with a history of hypertension as this will result in renal hypoperfusion)
- Sepsis
- High Early Warning Score
- Hypovolaemia

Prevention

Aim to:

- Identify patients at risk (see AKI risk factors)
- Optimise volume status
- Treat sepsis promptly
- Avoid nephrotoxins
- Review medications, e.g. adjust drug doses, withhold antihypertensives if hypotensive
- Reduce risk of contrast-induced AKI:
- The risk of contrast-induced AKI is small in the general population
- Patients at high risk are the acutely ill, with AKI/CKD, sepsis and hypovolaemia
- Consult the "Prevention of Contrast-Induced Acute Kidney Injury" guideline at www.renal.org

Staging and Classification

Acute kidney injury is defined as:

increase in serum creatinine of 26 µmol/L within 48 hours
OR
an increase in serum creatinine ≥1.5 times above baseline value within 1 week
OR
urine output of <0.5 ml/kg/hr for > 6 consecutive hours

If a baseline serum creatinine value is not available within 3 months and AKI is suspected:

- repeat serum creatinine within 24 hours
- a baseline serum creatinine value can be estimated from the nadir serum creatinine value if patient recovers from AKI

Patients that meet the definition for AKI can be staged according to whichever criteria (serum creatinine or urine output) gives them the highest stage.

KDIGO staging system for acute kidney injury:

Stage	Serum creatinine	Urine output
1	rise ≥26 µmol/L within 48 hr or rise ≥1.5 to 1.9 X baseline SCr	<0.5 mL/kg/hr for >6 consecutive hr
2	rise ≥2 to 2.9 X baseline SCr	<0.5 mL/kg/hr for >12 hr
3	rise ≥3 X baseline SCr or rise ≥ 354 µmol/L or Commenced on renal replacement therapy (RRT) irrespective of stage	<0.3 mL/kg/hr for >24 hr or anuria for 12 hr

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Assessment

History

Focus history on ascertaining the main causes of AKI (many patients have more than one cause of AKI):

Common causes

- Sepsis
- Hypoperfusion (hypotension, hypovolaemia, etc.)
- Medications
- Obstruction

Less common causes

(consider these if the common causes listed above are not obvious)

- Intrinsic renal diseases
 - Glomerulonephritis
 - Vasculitis
 - Interstitial nephritis
 - Myeloma

Remember that there are many causes of AKI:

Pre-renal (functional)

- Hypovolemia, e.g. bleeding, gastrointestinal losses
- Sepsis
- Cardiac arrhythmias
- Myocardial infarction
- Renal artery stenosis

Intrinsic renal (damage)

- Prolonged hypoperfusion causing tubular injury
- Infiltrative disease, e.g. myeloma
- Nephrotoxins
- Glomerulonephritis
- Interstitial nephritis
- Rhabdomyolysis

Post-renal (functional)

- Renal stone disease
- Pelvic masses, e.g. cervical cancer
- Prostatic hypertrophy / cancer
- Urethral stricture

Clues from the history

Sepsis

- Fever
- Cough with sputum
- Vomiting and diarrhoea
- Dysuria
- Urinary catheter
- Immunosuppression (can predispose and also prevent rise in white cell count masking sepsis)

Hypoperfusion

- Vomiting and/or diarrhoea
- Haemorrhage
- Cardiac failure (acute or chronic)
- Cardiac arrhythmias
- Diuretics (over diuresis)

Medications

- Non-steroidal anti-inflammatory drugs (NSAIDs are nephrotoxic and can also cause interstitial nephritis)
- Angiotensin-converting enzyme inhibitors (ACEi reduces renal blood flow)
- Angiotensin receptor blockers (ARBs reduces renal blood flow)
- Gentamicin and vancomycin (high levels of aminoglycosides are nephrotoxic)
- Iodinated contrast agents (contrast-induced AKI)
- Any new medication (some drugs can cause interstitial nephritis, e.g. NSAIDs, proton pump inhibitors and antibiotics)
- Herbal remedies (may contain nephrotoxic compounds)
- Over the counter medications

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Assessment

Clues from the history (cont.)

Obstruction

- History of kidney stones
- Prostatic symptoms (prostatic hypertrophy or malignancy)
- Known single kidney (obstruction of one ureter will cause AKI)
- Pelvic malignancy

Intrinsic renal disease

- Constitutional symptoms (fever and weight loss are non-specific but are features of vasculitis)
- Joint pains (vasculitis)
- Rashes (purpuric rashes or nodules may be a sign of vasculitis)
- Nasal stuffiness (sinus involvement in ANCA-associated vasculitis, e.g. granulomatosis with polyangiitis)
- Haemoptysis (vasculitis)
- Red eye (uveitis in vasculitis)
- Back pain (bone pain may be a sign of myeloma – the lower back is a common site)
- Falls/immobility (rhabdomyolysis)
- Neuropathies (vasculitis)



Typical vasculitic rash

Examination

Aim to identify clues as to the cause, assess volume status and identify any complications.

Identify the cause:

Pre-renal

- Volume status examination
- Evidence of sepsis, e.g. fever, respiratory signs, surgical site, red swollen joints, cellulitis, indwelling urinary catheters, cannulae
- Evidence of haemorrhage, e.g. haematemesis, melaena

Intrinsic

- Volume status examination
- Rash (vasculitis, interstitial nephritis)
- Red eye (vasculitis)
- Red swollen joints (vasculitis)
- Swollen limb (rhabdomyolysis)



Red eye may occur in vasculitis

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Assessment

Examination (cont.)

Post-renal

- Tense ascites (intra-abdominal hypertension)
- Palpable bladder
- Large prostate

Fluid status

- Capillary refill (<3 s)
- Pulse rate (tachycardia may be masked by beta blockers)
- Blood pressure (lying and standing if patient able to stand safely)
- Skin turgor (over clavicle)
- Jugular venous pressure
- Oedema (peripheral and pulmonary)
- Fluid balance charts
- Daily weights (trends in body weight are a sensitive indicator of volume status)

Complications

- Hyperkalaemia (potassium > 6.0 mmol/L)
- Acidosis
- Acute confusion (uraemic encephalopathy)
- Pulmonary oedema
- Pericarditis (pericardial friction rub)



Pulmonary oedema

Investigation

Aims to identify the cause and identify complications.

All patients:

- Biochemistry profile:
 - Urea
 - Liver function tests
 - Electrolytes
 - Glucose
 - Creatinine
 - Bone profile
 - Bicarbonate
- Full blood count
 - Look for evidence of haemorrhage and sepsis
 - Low platelets may occur with sepsis or haemolytic uraemic syndrome/thrombotic thrombocytopenic purpura
- Urinalysis
 - Blood and/or protein is abnormal
 - Urinary tract infection and vasculitis must be considered

Consider:

- Cultures:
 - blood
 - urine
 - wound
- C-reactive protein (CRP)
- Lactate (surrogate marker of shock and hypoperfusion)
- Venous or arterial blood gas
- Coagulation screen
- Creatine kinase (rhabdomyolysis)
- Lactate dehydrogenase (LDH)
- Blood film (HUS/TTP)
- Serum and urine electrophoresis (myeloma)
- Serum free light chains (myeloma)
- Anti-glomerular basement membrane antibodies (Goodpastures disease)

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Response

Investigation (cont.)

Consider:

- Urine Bence-Jones protein (myeloma)
- Anti-neutrophil cytoplasmic antibody (ANCA; vasculitis)
- Anti-nuclear antibody (ds DNA specific for SLE)
- Complement (C3 and 4)
- Hepatitis B and C serology
- Renal tract ultrasound (if no obvious precipitating cause and AKI not responding to supportive measures to check for renal anatomy and exclude obstruction)
- CXR (infection, pulmonary oedema, pulmonary haemorrhage)
- ECG (hyperkalaemia, pericarditis, arrhythmia, ischaemia)



Pneumonia

Management

Treatment is initially supportive but ultimately dependent upon the underlying cause. It is important to reduce both the severity and duration of AKI as this predicts progression to chronic kidney disease (CKD).

All patients:

Immediate assessment and response - ABCDE approach

1. Rapid correction of hypovolaemia and restoration of haemodynamic status

- 500 ml (250 ml if patient has a history of cardiac failure) of a balanced crystalloid stat (e.g. Hartmann's solution) rapidly
- If hyperkalaemia is present ($K^+ > 5.5$ mmol/L) in the setting of oliguric AKI or rhabdomyolysis 0.9% sodium chloride is preferred initially
- Conversion to a balanced crystalloid can be considered once potassium levels are known and good urine output established.
- Assess clinical response to fluid in terms of:
 - capillary refill time
 - pulse (reduction in pulse if tachycardic)
 - jugular venous pressure (rise in JVP)
 - blood pressure (rise in BP)
 - pulmonary oedema
 - urine output (increasing if oliguric)
- If no clinical response and no pulmonary oedema administer further 500 ml of crystalloid, reassess clinically and discuss with senior member of team
- If clinical response to fluid bolus continue with IV fluids and discuss further fluid therapy management plan with senior member of team
- If the patient develops oliguric AKI despite adequate volume resuscitation consider the patient as having volume unresponsive AKI. Further excessive fluid resuscitation may result in pulmonary oedema.
- If the patient has volume unresponsive AKI continue with IV fluid cautiously, matching urine output and monitoring for signs of respiratory distress (rising respiratory rate, pulmonary oedema or falling oxygen saturations)
- If the patient remains hypotensive despite volume resuscitation, vasopressor support may be required

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Response

Management (cont.)

- Prompt treatment of sepsis – ideally following appropriate cultures
 - Avoid nephrotoxins
 - Aminoglycosides
 - Iodinated contrast
 - Amphotericin
 - NSAIDs
 - Adjust dose of renally excreted drugs
 - Digoxin
 - Low molecular weight heparins
 - Opioids
 - Penicillin
 - Nutrition - seek dietician support
 - Referral
- Not all patients with AKI will need referral to the renal team.

Referral criteria to nephrology:

- AKI stage 3 ($SCr \geq 3 \times$ baseline value)
- Persistent oliguria and/or rising serum creatinine despite supportive therapy
- Complications refractory to medical treatment:
- Hyperkalaemia ($K^+ > 6.0$ mmol/L)
- Pulmonary oedema
- Acidosis ($pH < 7.15$)
- Uraemic encephalopathy
- Uraemic pericarditis

AKI plus:

- Absence of defined cause, e.g. sepsis, hypovolaemia
- Systemic features, e.g. rash, joint pains, blood and protein on urinalysis
- Paraprotein (myeloma)
- Haemolysis and low platelets (HUS/TTP)
- Poisoning suspected (ethylene glycol, methanol, lithium)

Hyperkalaemia

Symptoms:

- Usually asymptomatic
- Weakness, paraesthesiae and palpitations

ECG:

- May be normal even in life-threatening hyperkalaemia
- Peaked T waves
- Prolonged PR interval and eventual loss of P wave
- QRS widening
- AV dissociation
- Ventricular fibrillation or tachycardia

Management:

It must be remembered that unless the cause of the AKI is treated, the measures described are only temporary. The potassium will need to be monitored closely until recovery of sufficient kidney function to excrete potassium or renal replacement therapy is commenced.

Immediate treatment required if:

- $K^+ > 6.5$ mmol/L
- or
- ECG changes

Immediate treatment:

- IV 10 ml 10% calcium gluconate over 2-5 minutes (cautiously, as extravasation can cause tissue damage). This stabilises the myocardium rapidly, but has no effect on serum potassium concentration. Further doses may be required until reduction in plasma potassium concentration is achieved. Onset of action 2-4 minutes. Duration of action 30-60 minutes.

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Response

Hyperkalaemia (cont.)

Further treatment:

- 10U fast acting insulin (actrapid) added to 50 ml of 50% dextrose infused IV over 20 minutes to increase cellular potassium uptake. Blood glucose must be monitored closely to avoid hypoglycaemia. Onset of action 15-30 minutes. Duration of action 4-6 hours.
- Salbutamol 10-20 mg (5 mg back to back) nebuliser 6 hourly (caution if tachycardia or ischaemic heart disease) to stimulate cellular potassium uptake. Avoid in patients on beta blockers and/or who have a history of cardiac arrhythmias. Onset of action 30 minutes. Duration of action 2-4 hours.
- Medication review - stop any drugs that contain potassium or interfere with renal excretion of potassium (ACE inhibitors, angiotensin receptor blockers, beta blockers, potassium sparing diuretics)
- Review potassium intake including intravenous fluids and enteral or parenteral feeds.

For more details on hyperkalaemia management, see the Renal Association Guidelines at www.renal.org.

Recovery and follow up

The first signs of recovery from AKI may be

- an increase in urine output if the patient was oliguric and/or
- a reduction in the rise in the daily serum creatinine followed by a plateau in its value prior to a fall

Recovery from AKI can result in a polyuric state in some patients with the production of large urine volumes until the capacity of the renal tubule to concentrate urine returns. There must therefore be careful attention to the patient's volume status and fluid requirements.

Patients can be at risk of developing:

- Hypernatraemia - secondary to a free water deficit and requires an increased intake of water (intravenous 5% glucose if unable to take water orally). Failure to address the free water deficit promptly will not only slow renal recovery but will also put the patient at risk of neurological complications.
- Hypokalaemia - which requires appropriate therapy due to the risk of cardiac arrhythmias and ileus.

Discharge and follow up after AKI:

- Patients who have had an episode of AKI are at risk of chronic kidney disease long term. The risk depends upon the severity of the episode of AKI
- Kidney function should be checked prior to discharge. Patients will require long-term follow up if there is evidence of chronic kidney disease (CKD)
- Medications should be reviewed prior to discharge with a plan to reintroduce medications that may have been held during the acute illness, e.g. antihypertensives, diuretics at an appropriate time. This may require an early follow up with the GP.
- Patients should be informed as to why they developed AKI and their risk factors.
- GP discharge letter should include
 - Severity of AKI
 - Cause of AKI
 - Risk factors for AKI
 - Discharge kidney function
 - Advice on whether medications need to be reviewed or reintroduced
- Refer patients to nephrology left with an estimated glomerular filtration rate (eGFR) < 30 mL/min/1.73m²

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Information

Links to other resources

- Acute Kidney Injury Resource Pack
- Renal Association guidelines
- Sepsis guidelines
- AKI Consensus Conference papers
- British National Formulary
- RenalMed
- EdRen
- Kidney Disease: Improving Global Outcomes (KDIGO)
- London Acute Kidney Injury Network
- Recognising and Responding to Acute Patient Illness and Deterioration
- The Yorkshire AKI Network

About us

1. This project was supported by a grant from NHS Kidney Care.
For more information, visit www.kidneycare.nhs.uk
2. The Royal College of Physicians of Edinburgh exists to promote the highest standards in medicine, to meet doctors' educational and professional needs and to promote public health.
For more information, visit www.rcpe.ac.uk

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Disclaimer

Every clinician is responsible for their actions and this application is to be used as a guide only. The app is applicable to most patients with acute kidney injury treated in the hospital setting but is not applicable to all patients, and clinical judgement is always required in applying general guidance to the management of an individual patient. Any information contained in this app does not replace or supersede other clinical guidelines applicable at the local level (e.g. Trust, Hospital or department level guidelines) or national guidelines (e.g. NICE or SIGN)

It is the responsibility of the licensed prescriber to double check the indications, contraindications, and dosages of the medications and treatments they prescribe.

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