Fact sheet
Acute kidney injury

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Definition

Acute kidney injury (AKI) is a term used to cover a spectrum of injury to the kidneys. This term now replaces acute renal failure (ARF) as it describes more accurately that kidney injury can occur before failure occurs.

AKI is characterised by a decline in renal function over hours or days that can result in failure to maintain fluid, electrolyte, and acid-base homeostasis.¹

NICE clinical guideline *Acute kidney injury: prevention, detection and management [CG169]* recommends defining acute kidney injury by any of the following criteria:

- a rise in serum creatinine of 26 micromol/litre or greater within 48 hours
- a 50 percent or greater rise in serum creatinine known or presumed to have occurred within the past seven days
- a fall in urine output to less than 0.5 mL/kg/hour for more than six hours in adults and more than eight hours in children and young people
- a 25 percent or greater fall in eGFR in children and young people within the past seven days.²

AKI can be staged based on severity, and increasing severity of AKI correlates with higher risk of worse outcomes.³ Kidney Disease: Improving Global Outcomes (KDIGO) has produced a *Clinical practice guideline for acute kidney injury* which includes AKI staging information. The information in the following table is taken from this guideline.⁴

<table>
<thead>
<tr>
<th>Stage</th>
<th>Serum creatinine</th>
<th>Urine output</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1.5 to 1.9 times baseline OR greater than or equal to 0.3 mg/dl (greater than or equal 26.5 mmol/litre) increase</td>
<td>less than 0.5 mL/kg/h for 6 to 12 hours</td>
</tr>
<tr>
<td>2</td>
<td>2.0 to 2.9 times baseline</td>
<td>less than 0.5 mL/kg/h for greater than or equal to 12 hours</td>
</tr>
<tr>
<td>3</td>
<td>3.0 times baseline OR increase in serum creatinine to greater than or equal 4.0 mg/dl (greater than or equal 353.6 mmol/litre) OR initiation of renal replacement therapy OR, in patients less than 18 years, decrease in eGFR to less than 35 mL/min/1.73 m²</td>
<td>less than 0.3 mL/kg/h for greater than or equal 24 hours OR anuria (absence of urine production) for greater than or equal 12 hours</td>
</tr>
</tbody>
</table>

*Think Kidneys* is an NHS campaign to reduce avoidable harm and death to patients with AKI, and is led by NHS England in partnership with the UK renal registry.
In the following Think Kidneys video, Richard Fluck, the national clinical director for renal disease for NHS England, speaks about AKI and the Think Kidneys campaign.

**Think Kidneys video**

Prevalence and incidence

Kidney Care UK reports that:

- AKI affects 1 in 5 people admitted to hospital as an emergency and may be more deadly than a heart attack.
- In the UK, around 100,000 deaths each year are associated with AKI; that's equivalent to ten people every hour. Research shows that 30 percent of these could be prevented with the right care and treatment.
- The costs to the NHS of AKI are estimated to be between £434 million and £620 million per year, which is more than the costs associated with breast cancer, or lung and skin cancer combined.\(^5\)

AKI is commonly associated with acute illness. It is reported that more than 15 percent of those admitted to hospital in an emergency develop stage 1 AKI.\(^5\) Incidence of AKI is also increasing, potentially as a result of the ageing population in the UK with increasing comorbidities. Better detection methods may also play a part in the increase in reported incidence of AKI.\(^5\)

Signs and symptoms

In the early stages, AKI may be symptomless. Some people may produce less urine than usual but this isn’t always the case.

The following symptoms can develop rapidly if someone with AKI deteriorates:

- nausea and vomiting or diarrhoea, evidence of dehydration
- reduced urine output or changes to urine colour
- new or worsening confusion, fatigue and drowsiness.\(^6\)
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For a personal story about AKI, read Think Kidneys’ *Michael’s story – a patient’s experience of acute kidney injury.*

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**Causes/risk factors**
The causes of AKI can be divided into three categories, although more than one cause is often present.7

**Pre renal**
Pre-renal causes are the most common type of AKI. A pre-renal AKI is a result of reduced blood flow to the kidneys. Reduced blood flow can be caused by hypovolaemia (low blood volume), reduced cardiac output, or hypotension. Medicines may cause a pre-renal AKI by reducing blood pressure, circulating volume (eg, loop diuretics), or by affecting renal blood flow (eg, angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARBs) and non-steroidal anti-inflammatory drugs (NSAIDs)).7 A pre-renal AKI can also be caused by systemic vasodilation, for example in sepsis.8

**Intrinsic renal (or intrarenal)**
This is due to structural damage to the kidney tissues and may be a result of persistent pre or post-renal causes. Damage may also be caused by medicines such as antibiotics, X-ray contrast media or chemotherapy. Types of structural damage may be categorised as vascular, glomerular, tubular or interstitial depending on which area of the kidneys are affected.7

**Post-renal**
This is the least common cause of AKI (accounting for around 10 percent of AKIs) and is due to obstruction of the flow of urine out of the kidneys. This can be caused by renal stones, blocked catheters, an enlarged prostate or genitourinary masses.7

**Risk factors** for AKI include:
- aged over 65 years
- history of AKI
- **chronic kidney disease**
- symptoms or history of urological obstruction
- chronic conditions such as heart failure, liver disease, and diabetes mellitus
- neurological or cognitive impairment or disability (which may limit fluid intake because of reliance on a carer)
- **sepsis**
- hypovolaemia (low blood volume)
- oliguria (urine output less than 0.5 mL/kg/hour).
- use of medicines that negatively impact on the kidneys within the last week (especially if hypovolaemic), for example NSAIDs, ACE inhibitors, ARBs, and diuretics
- exposure to iodinated contrast agents (used in radiography) within the past week
- **cancer** and cancer therapy (risk will depend on the type of cancer, proposed treatment and premorbid risk factors)
- immunodeficiency, for example human immunodeficiency virus (HIV) infection
- toxins such as some herbal remedies, poisonous plants and animals.9,10
Think Kidneys has produced *Communities at risk of developing acute kidney injury*, which gives detailed information about patient groups who are at risk of AKI in both secondary and primary care. This document can be found on their *Pharmacists* page.

**Pathophysiology (mechanism of disease)**
For an introduction to the pathology of pre-renal, post-renal and intrarenal AKI, watch the following video.

*PhysioPathoPharmaco - Acute Kidney Injury (AKI): Prerenal, Intrarenal, Postrenal*

Please note that in this video the term ‘nephrotoxic’ is used. Think Kidneys recommends *the term “nephrotoxic” should be used with caution. Few medications truly have direct toxic effects on the kidneys, but several have the potential to impair renal function if used under certain circumstances, such as where the patient has a degree of chronic kidney disease in conjunction with hypovolaemia and acute illness. Under these circumstances, continued use of these medications may further exacerbate an episode of AKI.*

For more detailed information on the pathophysiology of AKI, access the following *Comprehensive Physiology* article *Pathophysiology of acute kidney injury.*

**Prognosis and complications**
Kidney Disease Improving Global Outcomes (KDIGO) guidance states that *the kidney is a fairly robust organ that can tolerate exposure to several insults without suffering significant structural or functional change. For this reason, any acute change in kidney function often indicates severe systemic derangement and predicts a poor prognosis.*

The prognosis of AKI varies depending on clinical setting, the underlying cause, and any comorbidities, and it’s been shown that early detection is likely to improve prognosis.

Complications of AKI can be serious and include electrolyte imbalances (including potentially serious hyperkalaemia), metabolic acidosis, volume overload, uraemia (raised urea and other nitrogenous waste)
and chronic kidney disease and end-stage renal disease. For more information about these complications, visit NICE clinical knowledge summary (CKS) Acute kidney injury - Complications.

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**Diagnosis/detection**

AKI is detected based on clinical assessment of signs and symptoms, risk factors, urine output and serum creatinine levels. Early detection and treatment of AKI may improve outcomes. Given that approximately two thirds of AKI cases begin in the community, those working in primary care have a crucial role to play in prevention, early detection and management, as well as post-AKI care.

**AKI warning stage test result**

The Acute kidney injury (AKI) algorithm endorsed by NHS England is used by laboratories to identify potential cases of AKI based on creatinine levels. An AKI warning stage test result is then communicated to GP clinical systems. This allows the primary care team to take action based on their clinical judgement.

To learn more about AKI warning stage test results, you can watch the following video:

*Think Kidneys*

Think Kidneys have also produced Acute kidney injury best practice guidance: Responding to AKI warning stage test results for adults in primary care which includes the following quick guides:

- **Table 1. Acute kidney injury: recommended response times to AKI warning stage test results for adults in primary care**
- **Table 2: Recognising and responding to acute kidney injury for adults in primary care**

The Royal College of General Practitioners (RCGP) has produced the Acute kidney injury toolkit which complements the Think Kidneys guidance, and includes information on recognising and responding to AKI warning stage test results for adults in primary care under Recognition and Response.

**NICE guidance on prevention, detection and management**

NICE clinical guideline Acute kidney injury: prevention, detection and management [CG169] outlines the recommendations for assessment of risk of AKI (Section 1.1 Assessing risk of acute kidney injury), detection of AKI (Section 1.3 Detecting acute kidney injury) and identifying causes of AKI (Section 1.4 Identifying the cause(s) of acute kidney injury).
Interpreting biochemistry information
To work through a biochemistry case study which looks at a person who experiences AKI, visit CPPE’s Biochemistry gateway page. Further information on interpretation of biochemistry data can also be found in the Further reading section of this gateway page.

Prevention
Prevention of AKI remains a national priority. Think Kidneys provides information for the public on how to keep their kidneys healthy. They offer an educational video, written information and a series of posters which can be found on their Think Kidneys – the national campaign to raise awareness of our kidneys – their importance for life and health and how to look after them page.

The NICE CKS Acute kidney injury - Scenario: Prevention of acute kidney injury covers prevention of AKI.

Prevention in older people and those living in care homes
Think Kidneys has developed practical resources to raise awareness and help with the prevention, detection and management of AKI in care homes on their Care homes webpage. This includes the learning guide Care homes: Acute kidney injury and hydration guide which contains example care plans in Appendix 3 (page 20).

The Oxford Academic Health Science Network have produced a video series about the importance of good hydration in keeping care home residents happy and healthy.

Oxford Academic Health Science Network - Good hydration! – improving hydration for care home residents

Management of AKI is dependent on the staging and presence of complicating factors.

Read Section 1.5 Managing acute kidney injury of Acute kidney injury: prevention, detection and management [CG169] for information about the management of those who develop AKI.
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All people who develop AKI should have a thorough review of their medicines to eliminate the potential cause or contributory factor for AKI, and ensure all medicines are clinically appropriate. Think Kidneys has produced *Guidelines for medicines optimisation in patients with acute kidney injury* to aid this medication review process.

In many cases of AKI, it may be necessary to withhold medicines used to treat cardiac conditions such as ACE inhibitors, ARBs, and diuretics. As a pharmacy professional you can help to ensure that medicines get reintroduced and re-titrated as appropriate following an AKI. In patients with heart failure with left ventricular systolic dysfunction, it may be necessary to refer to the specialist heart failure team for advice.

More information about managing these types of medicines can be found in the following documents:

- *Changes in kidney function and serum potassium during ACEI/ARB/diuretic treatment in primary care*
- Think Kidneys *When or if to re-start ACEI, ARB, diuretics and other antihypertensive drugs after an episode of acute kidney injury*

**Patient support**

Think Kidneys has an *Information for the public* page with links to support organisation. In addition to offering *resources for pharmacists* and *care homes*, Think Kidneys offer general advice for those working in *primary care* and *secondary care*. These pages link to patient advice leaflets.

**Further resources**

*KDIGO Clinical practice guideline for acute kidney injury* aims to assist practitioners caring for adults and children at risk for or with AKI, and contains chapters on definition, risk assessment, evaluation, prevention, and treatment.

CPPE *Fundamentals of renal therapeutics* e-course aims to develop pharmacy professionals’ knowledge and skills in renal therapeutics.

**External websites**

CPPE is not responsible for the content of any non-CPPE websites mentioned on this page or for the accuracy of any information to be found there.

All web links were accessed on 27 January 2020.
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