Acute Kidney Injury (AKI) In Primary Care

Supporting early detection and consistent management

Responding to AKI Warning Stage Test Results for Adults in Primary Care: Best Practice Guidance
AKI in Primary Care

AKI: Context and focus for primary care

- Definition, staging and association with acute illness
- Implications for patients, the NHS and primary care

AKI: Detection in primary care

- Identifying patients at risk of AKI
- Interpreting AKI warning stage test results within clinical context

AKI: Management in primary care

- Think Kidneys → Think Cause, Think Drugs, Think Fluid Status, Think Review
- When to consider admission and / or renal referral
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AKI: Context and focus for primary care

What is AKI?

➢ A sudden reduction in kidney function (► usually coincides with onset of acute illness)

Why is AKI important?

➢ Associated with adverse outcomes for patients (► consider AKI an acute illness severity marker)

➢ Common (► more than 1/2 million people in England develop AKI every year)

Why has a national “Think Kidneys” campaign been established to raise AKI awareness?

➢ Public¹ and Healthcare Professional² awareness of AKI is poor

➢ UK Study (2009) found deficiencies in AKI care were common - including delayed AKI recognition³

¹Ipsos MORI Survey (2014). Understanding what the public know about their kidneys and what they do.
Why are primary care teams being alerted to AKI?

- Many patients in community are at risk of AKI (► require prompt review when acutely unwell)
- Most AKI occurs in community (► 2/3 of hospital AKI cases begin pre-hospital admission)¹

What can primary care teams do to reduce patient harm caused by AKI?

1. Raise AKI awareness and limit AKI risk (► AKI often asymptomatic → further delaying AKI detection)
2. Promote prompt AKI detection (► consider AKI early during acute illness episodes)
3. Initiate simple interventions early (► increase chance of recovery / reduce treatment costs)
4. Perform post-AKI review (► detect new or progressive CKD +/- restart drugs suspended during AKI)

¹ Selby et al. (2012). Defining the Cause of Death in Hospitalised Patients with AKI. PLoS ONE. 7 (11): e48580
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AKI: Definition, Staging and Association with Acute Illness

AKI definition

- AKI is a clinical and biochemical syndrome reflecting abrupt kidney dysfunction
- AKI is not a primary disease nor a “diagnosis”
- AKI is a heterogeneous syndrome with various causes and variable outcomes

AKI staging

- AKI stage is determined by acute changes to serum creatinine and/or urine output
- AKI usually occurs secondary to acute illness (commonly sepsis)
- Identifying underlying acute illness causing AKI is key to establishing primary diagnosis
- Treating underlying acute illness key to treating most AKI
# AKI Definition (Kidney Disease Improving Global Outcomes, KDIGO criteria)

<table>
<thead>
<tr>
<th>AKI Definition</th>
<th>Serum Creatinine&lt;sup&gt;2&lt;/sup&gt;</th>
<th>Urine Output&lt;sup&gt;4&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increase in serum creatinine by &gt;26(\mu)mol/L ≤ 48 hrs</td>
<td></td>
<td>Urine volume &lt;0.5 mL/kg/hr for ≥ 6 hrs</td>
</tr>
<tr>
<td>Increase in serum creatinine by ≥ 1.5 times baseline&lt;sup&gt;3&lt;/sup&gt; which is known or presumed to have occurred within previous 7 days</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<sup>2</sup>Note serum creatinine changes and **not estimated** GFR (eGFR) define AKI (as eGFR is **not** a reliable indicator of true GFR during unsteady clinical states associated with AKI) ► **Drug dosing** should **not** be based upon eGFR during AKI episodes.

<sup>3</sup>‘Baseline’ creatinine value should be considered as the patient’s ‘usual’ creatinine when clinically well → determine by reviewing patient’s previous blood results within clinical context. Assume normal baseline if no previous blood tests.

<sup>4</sup>In practice **urine output criteria** can only be applied to hospitalised patients who are catheterised - **but a reliable history of low or absent urine output should alert the clinician to the possibility of AKI.**

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<table>
<thead>
<tr>
<th>AKI Stage</th>
<th>Serum Creatinine</th>
<th>Urine Output</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 1</td>
<td>Increase in serum creatinine by &gt;26µmol/L ≤ 48 hrs OR an increase in serum creatinine by ≥ 1.5 x baseline</td>
<td>urine output &lt;0.5mL/kg/hr for 6-12hrs</td>
</tr>
<tr>
<td>Stage 2</td>
<td>Increase in serum creatinine by ≥ 2 x baseline</td>
<td>urine output &lt;0.5mL/kg/h for ≥12hrs</td>
</tr>
<tr>
<td>Stage 3</td>
<td>Increase in serum creatinine by ≥ 3 x baseline OR an increase in serum creatinine by ≥1.5 baseline to &gt; 354 µmol/L</td>
<td>urine output &lt;0.3mL/kg/h for ≥24hrs OR anuria for ≥12 h</td>
</tr>
</tbody>
</table>

2 When creatinine change is known or presumed to have occurred within previous 7 days

AKI as a patient safety barometer associated with acute illness

Prompt recognition and good management of AKI requires and often reflects prompt recognition and good management of acutely unwell patients.
AKI in primary care

**AKI: Context and focus**

- Definition, staging and association with acute illness
- Implications for patients, the NHS and primary care

**AKI: Detection in primary care**

- Identifying patients at risk of AKI
- Interpreting AKI warning stage test results within clinical context

**AKI: Management in primary care**

- Think Kidneys → Think **Cause**, Think **Drugs**, Think **Fluid Status**, Think **Review**
- When to consider admission and / or renal referral
AKI Patient Implications: Independently associated with adverse acute and chronic outcomes

- **AKI associated with increased patient mortality**
- **Odds of death ∝ AKI severity** in UK Study

![Graph showing mortality percentages across stages of AKI and non-elective mortality](image)

- Other studies show association with death persists if
  - Acute and chronic co-morbidities accounted for
  - Patients followed up post discharge / longer term

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AKI Patient Implications: Independently associated with adverse acute and chronic outcomes

- AKI associated with increased patient morbidity
- Meta-analysis shows AKI is risk factor for CKD

![Graph showing pooled hazard adjusted ratios for CKD post-AKI.](image)

Pooled hazard adjusted ratios for CKD post-AKI

- CKD also associated with ↑ risk of end-stage renal failure, cardiovascular disease and death

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AKI NHS Implications: Significant additional impact on Healthcare Resources

- AKI commonly complicates acute illness and hospital admissions
- AKI associated with 25.4% of unselected emergency admissions to a large UK acute hospital Trust\(^1\)
- AKI increases duration and complexity of acute illness ▶ AKI increases length of hospital stay (LOS)\(^1\):
  - AKI group LOS almost 3x higher than non AKI group (10 vs 4 days)\(^1\)
  - AKI group more often required critical care beds (8.1% vs 1.7%)\(^1\)
- AKI associated with complex treatments such as dialysis (▶ may be required permanently)
- AKI significantly increases healthcare costs as a consequence of these complications

\(^1\) Challiner et al. (2014). Incidence and consequence of AKI in unselected emergency admissions to a large acute UK hospital trust. BMC Nephrology. 15:84
AKI aspirations for primary care teams

Primary care teams **well located** to:

1. **Raise AKI awareness** and **limit AKI risk** in “at risk” patient groups
2. **Detect AKI** and deliver **simple interventions early** (to limit AKI severity and duration)
3. **Undertake post AKI review** to
   a. Detect new or worsening **Chronic Kidney Disease** post AKI
   b. Restart drugs suspended during AKI (especially if **prognostic** benefit)
   c. Limit risk of further AKI (patient / carer advice where appropriate)
AKI and primary care: Prompt detection and management

Two National AKI Patient Safety Alerts aim to promote AKI care in the community

- Mandates national automated AKI detection system to generate AKI warning alerts alongside blood tests
- Pilot studies indicate Full Time Equivalent GP expects about one AKI e-alert every 1-2 months (>\frac{1}{2} likely AKI Stage 1)

- Health care staff should be signposted to Think Kidneys AKI resources (hyperlinks to relevant resources at foot of slides)
- Resources include AKI guidelines to support appropriate response to AKI warning alerts by Primary Care Teams

THINK KIDNEYS Resource: Full Primary Care AKI Guidelines [LINK]
AKI also associated with **adverse long term outcomes**

- **Renal Health**: AKI is associated with **new** or **worsening** CKD, including ESRF
  - especially if **severe** or **multi-hit** AKI in
    1. Elderly patients
    2. Patients with **diabetes**
    3. Patients with **pre-existing** CKD

- **General Health**: Drugs with **prognostic** long term benefit (e.g., ACE-I for heart failure) may be suspended in **clinical context** of acute illness and AKI
  - **long term prognostic benefit** of such drugs **lost** if not restarted post AKI

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**Pooled hazard adjusted ratios for End-Stage Renal Failure (ESRF) post-AKI**

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AKI and Primary Care: Post AKI review

Post AKI reviews consider:

1. Monitoring for new or worsening CKD especially if creatinine has not returned to baseline or other CKD risk factors. NICE guidelines advocate monitoring renal function for 3 years post AKI.

2. Restarting drugs with prognostic benefit once clinical context improves and stabilises / acute illness resolved unless compelling contraindication to drug remains (or ongoing AKI risk > drug benefit).

3. Onward drug adjustments tailored to chronic disease and acute clinical context (see next 2 slides)

4. Patient / carer advice to limit further AKI episodes (utilise AKI Patient Leaflets, links below)
   - Encourage early medical contact to assess blood pressure, renal function and medications if
     a. Acutely unwell
     b. Unable to maintain good fluid intake
     c. Reduced urine output noted

THINK KIDNEYS Resources: Restarting drugs LINK, Sick Day Guidance for Drugs LINK, Patient Advice Leaflets for those who have sustained AKI LINK and those at persistent risk of AKI LINK
Clinical Context and ACE-Inhibitors

Current Clinical Context

- Sepsis or other acute illness
- Hypovolaemia
- True or relative hypotension (+/- other anti-hypertensives if BP low)

During unstable context such drugs may
1. Worsen renal perfusion
2. Magnify AKI severity / duration

AKI Risk

Threshold to suspend ACE-I / ARB
(+/- other anti-hypertensives if BP low)

Long term ACE-I therapy of proven prognostic benefit in such patient groups

Heart Failure
Diabetes Mellitus
Chronic Kidney Disease

Chronic Clinical Context

Same patient groups at high risk of AKI if acutely unwell

Unstable Clinical Context

- Severe hypotension
- Hypovolaemia
- True or relative hypotension

Assess

Long term ACE-I therapy of proven prognostic benefit in such patient groups
Clinical Context and ACE-Inhibitors

Current Clinical Context

Stable Clinical Context
- Recovery from acute illness / clinically stable
- Restoration of volume / BP
- Post AKI / Renal function stabilised

Threshold to resume ACE-I / ARB
- Try to restart drug if strong indication
- Review patient & bloods ≤ 1-2 weeks
- See resource below but generally:
  - Initial creatinine rise ≤ 30% often OK
  - If creatinine rise ≥ 30% / progressive:
    1. Suspend drug and review patient
    2. Consider renal opinion
    3. If indication for ACE-I is heart failure
       consider cardiology opinion

Prognostic benefit of ACE-I / ARB

THINK KIDNEYS RESOURCE: ACE-Inhibitor and diuretic use in Primary Care
**ACE-I / ARB initiation or dose up-titration in Primary Care**

### Initial Assessment

1. Ensure clinical context is stable ➤ consider patient ‘sick day’ advice.
2. Use Immediate pre-treatment creatinine as baseline creatinine.
3. Arrange repeat blood tests within 1-2 weeks.

### Serum creatinine rise > 15% but < 30% from baseline

1. Continue drug but arrange to re-assess clinical status, BP and bloods within 1-2 weeks.
2. Consider ↓ other BP-lowering drugs if sBP <120, including diuretics if evidence of hypovolaemia.
3. Continue drug if creatinine stabilises on repeat testing (<30% above pre-treatment baseline).

### Serum creatinine rise > 30% from baseline

1. Promptly re-assess clinical, fluid and BP status.
2. Consider ↓ other BP-lowering drugs if sBP <120, including diuretics if evidence of hypovolaemia.
3. Repeat bloods ≤ 5-7 days ➤ if renal function remains >30% despite above measures:
   1. Stop drug and consider local renal opinion
   2. If indication for drug is heart failure also obtain advice from local heart failure team

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**THINK KIDNEYS RESOURCE:** ACE-Inhibitor and diuretic use in Primary Care [LINK]

**THINK KIDNEYS RESOURCE:** Patient Sick Day Guidance for Drugs [LINK]
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AKI in Primary Care: Patients at risk of AKI in Community

**Situation Specific - Exposure**
- Any acutely unwell patient is at acute risk of AKI
- ↑ AKI vigilance in clinically unstable, especially if
  - Hypovolaemia, dehydration, reduced oral intake
  - Absolute hypotension (sBP < 90 mmHg)
  - Relative hypotension (↓40 mmHg from baseline BP)
  - Sepsis
  - Recent operation or iodinated contrast scan
  - NSAIDs, BP-lowering ± diuretic drug use ≤ 1 week

**Patient Specific - Susceptibility**
- Many patients remain at persistent ↑ AKI risk
- ↑ AKI vigilance in ‘at risk’ communities
  - Older age patients (especially with polypharmacy)
  - Co-morbidities (eg. CKD, DM & Heart Failure)
  - Psycho-social setting (eg. In care home, ↓ mobility / dementia → unable to self regulate fluid intake)
- Consider risk reduction strategies in such groups
  - promote self care (or carers) to avoid dehydration
  - similar advice / resources as for "Post AKI review"

**THINK KIDNEYS Resource:** Advice on communities at risk of AKI [LINK]
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Interpreting AKI warning stage test results in Primary Care: Clinical context is Key

- A national automated AKI detection system aims to improve early recognition of AKI
- Presentation of AKI warning alerts depends upon Pathology System used, examples below:

<table>
<thead>
<tr>
<th>Sodium</th>
<th>125 mmol/L</th>
<th>(120 to 146)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Potassium</td>
<td>4.2 mmol/L</td>
<td>(3.5 to 5.3)</td>
</tr>
<tr>
<td>Bicarbonate</td>
<td>28 mmol/L</td>
<td>(22 to 29)</td>
</tr>
<tr>
<td>Chloride</td>
<td>87 mmol/L</td>
<td>(95 to 108)</td>
</tr>
<tr>
<td>Urea</td>
<td>25.6 mmol/L</td>
<td>(2.5 to 7.8)</td>
</tr>
<tr>
<td>Creatinine</td>
<td>611 umol/L</td>
<td>(50 to 120)</td>
</tr>
<tr>
<td>eGFR/1.73M²2</td>
<td>6 ml/min</td>
<td></td>
</tr>
<tr>
<td>AKI Warning stage</td>
<td>3</td>
<td></td>
</tr>
</tbody>
</table>

- Alert system relies upon computerised interpretation of blood results in isolation from clinical context
- AKI is not merely a ‘biochemical finding’ ► do not rely upon alert system to detect all AKI cases
- Always review current and previous blood results within clinical context in order to validate AKI alert
Interpreting AKI warning stage test results in Primary Care: Clinical context is Key

A positive AKI alert simply alerts clinician to possibility of AKI ► false positives can occur (see table below)

A negative AKI alert does not always rule out AKI ► false negatives can occur (see table below)

<table>
<thead>
<tr>
<th>False positive examples</th>
<th>False negatives example</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Recent Pregnancy:</strong> Creatinine falls during pregnancy</td>
<td><strong>Previous AKI within last year:</strong> Algorithm may calculate spursiously high baseline creatinine for patient.</td>
</tr>
<tr>
<td>► creatinine rise expected / normal post delivery.</td>
<td></td>
</tr>
<tr>
<td><strong>Drugs (eg. trimethoprim) inhibiting tubular creatinine secretion:</strong> Can cause creatinine rise whilst GFR stable.</td>
<td></td>
</tr>
<tr>
<td><strong>Recent IV fluid:</strong> ► spuriously low baseline creatinine.</td>
<td></td>
</tr>
</tbody>
</table>

If alert unexpected and stable clinical context ► consider repeating bloods within 48-72hrs to determine whether any creatinine changes are truly dynamic (AKI) or relatively stable / false positive.

If no alert issued though high clinical suspicion of AKI / acute illness ► it especially important for clinician to review current and previous blood results before ruling out AKI.

THINK KIDNEYS Resource: Further guidance on page 7 of Primary Care AKI Guidelines [Link]
Interpreting AKI warning stage test results in Primary Care: Infrastructure & Process

Any automated detection system is **only effective** if leads to **timely** and **appropriate intervention**

Detection system does **not issue interruptive alert** and in isolation does **not** ensure **timely intervention**

**Correct effective utilisation** of automated AKI system thus requires clinicians:

1. **Actively review alerts** within clinical context in **timely fashion**
   - Practices should ensure clinicians reviewing alerts know **reason why** blood tests were taken
   - **Particular challenge** if results reviewed by ‘out of hours’ services

2. **Respond to alerts** and clinical context with **timely intervention**
   - Think Kidneys Primary Care Resource includes **recommended alert response times** (►next slide)

**THINK KIDNEYS** Resource: Full Primary Care AKI Guidelines **LINK**
Table 1: Recommended response times to AKI Warning Stage Test Results for Adults in Primary Care

<table>
<thead>
<tr>
<th>AKI Warning Stage Test Result</th>
<th>LOW Pre-test Probability of AKI</th>
<th>HIGH Pre-test Probability of AKI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Confirm or refute automated AKI Test Result by comparing patient's current creatinine against patient's baseline creatinine</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Clinical Context Within Which Blood Test Taken</th>
<th>AKI Warning Stage 1</th>
<th>AKI Warning Stage 2</th>
<th>AKI Warning Stage 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>LOW Pre-test Probability of AKI</td>
<td>Current creatinine &gt; 1.5 x baseline level (or creatinine rise &gt; 26 μmol/L ≤ 48 hrs)</td>
<td>Current creatinine &gt; 2 x baseline level</td>
<td>Current creatinine &gt; 3 x baseline level (or creatinine &gt; 1.5 x baseline and &gt; 354 μmol/L)</td>
</tr>
<tr>
<td>Consider clinical review ≤ 72 hours of e-alert</td>
<td>Consider clinical review ≤ 24 hours of e-alert</td>
<td>Consider clinical review ≤ 24 hours of e-alert</td>
<td>Consider clinical review ≤ 6 hours of e-alert</td>
</tr>
<tr>
<td>If AKI confirmed</td>
<td>If AKI confirmed</td>
<td>If AKI confirmed</td>
<td>Consider immediate admission</td>
</tr>
<tr>
<td>Manage as per Table 2</td>
<td>Manage as per Table 2</td>
<td>Manage as per Table 2</td>
<td>Likely Stage 3 AKI</td>
</tr>
</tbody>
</table>

1. **What was clinical context when the blood test was taken?**
   - Stable Clinical Context
   - Unstable Clinical Context
     - Chronic disease / drug monitoring
     - Assessment of acute illness
     - (Assume unstable clinical context if clinical context unknown)

2. **Are risk factors for AKI present?**
   - Chronic AKI Risk Factors
   - Acute AKI Risk Factors
     - Chronic Kidney Disease
     - Acute illness
     - Chronic Heart Failure / Liver Disease
     - New drug started
     - Diabetes Mellitus
     - Poor oral fluid intake
     - Cognitive / Neurological Disease
     - Recent previous AKI

3. **Are factors present to suggest acute kidney dysfunction?**
   - Clinical Features
   - Biochemical Features
     - Reduced urine output
     - Creatinine rise from recent baseline
     - Patient unwell
     - Further creatinine rise on repeat test

4. **Are additional factors present to prompt early review?**
   - Patient Factors
   - Clinical / Biochemical factors
     - Stage 4 or 5 CKD
     - Patient unwell
     - Kidney transplant recipient
     - Serum K+ ≥ 6.0 mmol/l
     - Frail / co-morbidities
     - Likely intrinsic kidney disease
     - Urinary tract obstruction

Providing access to salient clinical data when taking blood tests via laboratory forms, medical records or handover will support timely appropriate response → especially when alert reviewed by out of hours GP unfamiliar with patient
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# Common causes of AKI in Primary Care: Pre-renal and Post Renal AKI

<table>
<thead>
<tr>
<th>AKI: Pre-renal Renal Insults</th>
<th>AKI: Post-renal Renal Insults</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Kidney function</strong> requires <strong>adequate renal perfusion</strong></td>
<td><strong>Kidney function</strong> requires <strong>adequate urine drainage</strong></td>
</tr>
<tr>
<td>80-90% of all AKI due to <strong>acute illness</strong> causing a significant / sustained reduction in renal perfusion:</td>
<td>↓ threshold for early renal USS in patients reporting ↓ urine output, especially if unwell +/- history of:</td>
</tr>
<tr>
<td>- Vasodilatation and <strong>hypotension</strong> due to sepsis</td>
<td>- Males with enlarged prostates</td>
</tr>
<tr>
<td>- ECV loss due to diarrhoea &amp; vomiting, or bleeding</td>
<td>- Renal calculi</td>
</tr>
<tr>
<td>- Hypotension due to <strong>acute heart failure</strong></td>
<td>- Pelvic or abdominal masses</td>
</tr>
<tr>
<td>Some <strong>drugs</strong> may magnify AKI during such states</td>
<td><strong>Delays</strong> relieving obstruction may magnify AKI</td>
</tr>
</tbody>
</table>

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**THINK KIDNEYS** Resource: [AKI & Drugs Guideline](#)
AKI Causes: Pre-renal and Post-renal account for majority of AKI

Addressing these 4 common drivers of AKI will address majority (> 90%) of AKI

Volume depletion

Drugs

Sepsis

Obstruction

Intrinsic kidney disease is a less common cause of AKI...

But important not to miss

MOST AKI
AKI Causes: When to suspect Intrinsic Renal Disease

- **Intrinsic Renal Disease** is a less common cause of AKI (≤ 5%)

- **Important not to miss**: may benefit from early renal referral

- Group of disorders reflecting toxin and/or immune-mediated kidney damage

- **Urine dip often key to diagnosis**: protein ± blood on dip should **raise suspicion** of intrinsic disease

- Myeloma and Tubulo-Interstitial Nephritis (TIN) exceptions: can be present with normal urine dip

- Especially **consider intrinsic renal disease** as cause of AKI if:

  1. No common / obvious cause for AKI (ie. sepsis, volume depletion, drugs or obstruction) and / or

  2. Urine dip +ve for protein +/- blood and / or

  3. Clinical features of nephritis or systemic disease causing AKI present (see next slide)
## AKI Causes: When to suspect Intrinsic Renal Disease

### Clinical Clues and Screening for intrinsic renal disease in AKI

<table>
<thead>
<tr>
<th>Clinical context</th>
<th>Potential diagnosis</th>
<th>Example screening tests</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rash +/- arthralgia</td>
<td>SLE, vasculitis, HSP, cryoglobulinaemia</td>
<td>ANA, ANCA, ↓ complement</td>
</tr>
<tr>
<td>Haemoptysis</td>
<td>Anti-GBM disease, vasculitis</td>
<td>Anti-GBM Ab, ANCA</td>
</tr>
<tr>
<td>Crush injury / long lie</td>
<td>Rhabdomyolysis</td>
<td>Creatine kinase</td>
</tr>
<tr>
<td>Haemolysis &amp; ↓ platelets</td>
<td>Thrombotic microangiopathy (TTP, HUS)</td>
<td>Blood film, LDH</td>
</tr>
<tr>
<td>Severe hypertension</td>
<td>Malignant hypertension</td>
<td>Fundoscopy</td>
</tr>
<tr>
<td>Vascular intervention</td>
<td>Cholesterol embolisation</td>
<td>↓ Complement, ↑ eosinophils</td>
</tr>
<tr>
<td>Recent chemotherapy</td>
<td>Tumour lysis syndrome</td>
<td>Uric acid level</td>
</tr>
<tr>
<td>↑ Ca²⁺ +/- bone pain</td>
<td>Multiple myeloma</td>
<td>Myeloma screen</td>
</tr>
<tr>
<td>Recently started new drug</td>
<td>Tubulo-Interstitial Nephritis (TIN)</td>
<td>↑ Eosinophils (not always)</td>
</tr>
</tbody>
</table>
**Table 2: Management of AKI in Primary Care**

<table>
<thead>
<tr>
<th>&quot;Think&quot; Cause</th>
<th>&quot;Think&quot; Medication</th>
<th>&quot;Think&quot; Fluids</th>
<th>&quot;Think&quot; Review</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Review patient within clinical context</strong></td>
<td><strong>Review drugs within clinical context</strong></td>
<td><strong>Tailor fluid advice to clinical context</strong></td>
<td><strong>Time next review to clinical &amp; chemical context</strong></td>
</tr>
<tr>
<td>History of acute illness?</td>
<td>Could drug be driving AKI?</td>
<td>If hypovolemic consider if</td>
<td>Consider early review (&lt; 12 hours) +/- admission if</td>
</tr>
<tr>
<td>➢ Think Sepsis</td>
<td>➢ Think suspend drug? eg</td>
<td>➢ Urine output +/- BP low?</td>
<td>➢ Patient unwell</td>
</tr>
<tr>
<td>➢ Think Hypotension</td>
<td>• NSAIDs</td>
<td>➢ Can patient drink more?</td>
<td>➢ Stage 3 AKI</td>
</tr>
<tr>
<td>Positive Urinalysis?</td>
<td>• BP drugs if low BP</td>
<td>➢ Are IV fluids required?</td>
<td>➢ K⁺ &gt;6.5 (not haemolysed)</td>
</tr>
<tr>
<td>UTI symptoms absent?</td>
<td>• Diuretics if dehydrated</td>
<td></td>
<td>➢ Risk of lung oedema</td>
</tr>
<tr>
<td>➢ Think intrinsic disease</td>
<td>Could drug accumulate?</td>
<td>If fluid overloaded consider</td>
<td>Consider repeating bloods:</td>
</tr>
<tr>
<td>Urinary Tract Symptoms?</td>
<td>➢ Think change dose? eg</td>
<td>➢ If risk of lung oedema?</td>
<td>➢ ≤ 72 hrs for stage 1 AKI</td>
</tr>
<tr>
<td>Palpable bladder?</td>
<td>• Diabetic medication</td>
<td>➢ Is patient passing urine?</td>
<td>➢ ≤ 24 hrs for stage 2 AKI</td>
</tr>
<tr>
<td>Consider urgent USS</td>
<td>• Digoxin</td>
<td>➢ Are diuretics indicated?</td>
<td>➢ ≤ 12 hrs for stage 3 AKI</td>
</tr>
<tr>
<td>➢ Think obstruction</td>
<td>• Opiates / gabapentin</td>
<td></td>
<td>and</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Ensure clinical context for repeat bloods handed over to those reviewing results</td>
</tr>
</tbody>
</table>
Contact Think Kidneys

How to find out more

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