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

Reviewed November 2018

Next review June 2021
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

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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
AKI in primary care

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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\(^1\))

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

\(^2\)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.

\(^2\) Note timescale of creatinine change is central to AKI definition → if no recent preceding blood test then incorporate clinical context to determine if creatinine change likely to have occurred during preceding week (ie. ‘acutely’).

\(^3\) ‘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.

\(^4\) 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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# AKI Staging (Kidney Disease Improving Global Outcomes, KDIGO criteria\(^1\))

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

\(^2\) When creatinine change is known or presumed to have occurred **within previous 7 days**

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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.

BIDIRECTIONAL RELATIONSHIP

- **Acutely Unwell Patient**
  - ↑ HR
  - ↑ CRP

- **Deteriorating Patient**
  - ↑ Temp
  - ↓ BP

- **AKI**
  - ↑ Creatinine
  - ↓ Urine output
AKI in primary care

AKI: Context and focus

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- Implications for patients, the NHS and primary care

AKI: Detection in primary care

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AKI: Management in primary care

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- 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 Study¹

![Mortality Bar Chart]

- **Other studies show association with death persists** if
  - Acute and chronic co-morbidities accounted for²
  - Patients followed up post discharge / longer term³

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¹ Selby N. et al. (2012). Use of Electronic Results Reporting to Diagnose and Monitor AKI in Hospitalized Patients. CJASN. 7:533-540.
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

Pooled hazard adjusted ratios for CKD post-AKI

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

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

- AKI increases duration and complexity of acute illness: AKI increases **length of hospital stay (LOS)**:
  - AKI group LOS **almost 3x higher** than non AKI group (10 vs 4 days)
  - AKI group more often **required critical care beds** (8.1% vs 1.7%)

- 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 and Primary Care: Post AKI review

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 (eg 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

**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 2 next 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

Heart Failure, Diabetes Mellitus, Chronic Kidney Disease

Chronic Clinical Context

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

Assess

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

Same patient groups at high risk of AKI if acutely unwell

Duraption
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 [LINK]
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

**Continuing ACE-I in chronic heart failure sometimes may be overall beneficial even if creatinine rise > 30%**

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>(121 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/1.73M²</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 ► creatinine rise expected / normal post delivery.</td>
<td><strong>Previous AKI within last year:</strong> Algorithm may calculate spuriously high baseline creatinine for patient.</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>Confirm or refute automated AKI Test Result by comparing patient's current creatinine against patient's baseline creatinine</th>
</tr>
</thead>
<tbody>
<tr>
<td>LOW Pre-test Probability of AKI</td>
<td>HIGH Pre-test Probability of AKI</td>
</tr>
<tr>
<td><strong>AKI Warning Stage 1</strong></td>
<td></td>
</tr>
<tr>
<td>Current creatinine &gt; 1.5 x baseline level (or creatinine rise &gt; 26 (\mu)mol/L (\leq 48) hrs)</td>
<td>Consider clinical review (\leq 72) hours of e-alert</td>
</tr>
<tr>
<td>If AKI confirmed (\rightarrow) manage as per table 2</td>
<td>Consider clinical review (\leq 24) hours of e-alert</td>
</tr>
<tr>
<td>Likely Stage 1 AKI (\rightarrow) manage as per table 2</td>
<td>Consider clinical review (\leq 6) hours of e-alert</td>
</tr>
<tr>
<td><strong>AKI Warning Stage 2</strong></td>
<td></td>
</tr>
<tr>
<td>Current creatinine &gt; 2 x baseline level</td>
<td>Consider clinical review (\leq 24) hours of e-alert</td>
</tr>
<tr>
<td>If AKI confirmed (\rightarrow) manage as per table 2</td>
<td>Consider clinical review (\leq 6) hours of e-alert</td>
</tr>
<tr>
<td>Likely Stage 2 AKI (\rightarrow) manage as per table 2</td>
<td></td>
</tr>
<tr>
<td><strong>AKI Warning Stage 3</strong></td>
<td></td>
</tr>
<tr>
<td>Current creatinine &gt; 3 x baseline level (or creatinine &gt; 1.5 x baseline and &gt; 354 (\mu)mol/L)</td>
<td>Consider clinical review (\leq 6) hours of e-alert</td>
</tr>
<tr>
<td>If AKI confirmed (\rightarrow) consider admission</td>
<td>Consider Immediate Admission</td>
</tr>
<tr>
<td>Likely Stage 3 AKI (\rightarrow) manage as per table 2</td>
<td></td>
</tr>
</tbody>
</table>

In order to utilise this table correctly first determine pre-test probability that creatinine rise reflects true AKI by considering:

1. **What was clinical context when the blood test was taken?**
   - Stable Clinical Context
   - Unstable Clinical Context
   - Clinical context when the blood test was taken (Assume unstable clinical context if clinical context unknown)

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

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

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

Providing access to salient clinical data when taking blood tests via laboratory forms, medical records or handover will support timely appropriate response \(\rightarrow\) 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

### AKI: Pre-renal Renal Insults
- **Kidney function** requires **adequate renal perfusion**
- **80-90%** of all AKI due to **acute illness** causing a significant / sustained reduction in renal perfusion:
  - Vasodilatation and **hypotension** due to sepsis
  - ECV loss due to diarrhea & vomiting, or **bleeding**
  - Hypotension due to **acute heart failure**
- Some **drugs** may magnify AKI during such states

### AKI: Post-renal Renal Insults
- **Kidney function** requires **adequate urine drainage**
- **↓** threshold for early renal USS in patients reporting **↓** urine output, especially if unwell +/- history of:
  - Males with enlarged prostates
  - Renal calculi
  - Pelvic or abdominal masses
- **Delays** relieving obstruction may magnify AKI

[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
Sepsis
Obstruction
Drugs

Intrinsic kidney disease is a less common cause of AKI...
But important not to miss
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 NSAIDs</td>
<td>➢ Urine output +/- BP low?</td>
<td>➢ Patient unwell</td>
</tr>
<tr>
<td>➢ Think Hypotension</td>
<td>➢ BP drugs if low BP</td>
<td>➢ Can patient drink more?</td>
<td>➢ Stage 3 AKI</td>
</tr>
<tr>
<td>➢ Think intrinsic disease</td>
<td>➢ Diuretics if dehydrated</td>
<td>➢ Are IV fluids required?</td>
<td>➢ K⁺ &gt;6.5 (not haemolysed)</td>
</tr>
<tr>
<td>UTI symptoms absent?</td>
<td>➢ Think change dose? eg Diabetic medication</td>
<td>➢ If risk of lung oedema?</td>
<td>Consider repeating bloods:</td>
</tr>
<tr>
<td>➢ Think intrinsic disease</td>
<td>➢ Diogoxin</td>
<td>➢ Is patient passing urine?</td>
<td>≤ 72 hrs for stage 1 AKI</td>
</tr>
<tr>
<td>Urinary Tract Symptoms?</td>
<td>➢ Opiates / gabapentin</td>
<td>➢ Are diuretics indicated?</td>
<td>≤ 24 hrs for stage 2 AKI</td>
</tr>
<tr>
<td>Palpable bladder?</td>
<td>Could new drug cause AKI?</td>
<td></td>
<td>≤ 12 hrs for stage 3 AKI</td>
</tr>
<tr>
<td>Consider urgent USS</td>
<td>➢ Think causes of TIN ? eg NSAIDs, antibiotics</td>
<td>and</td>
<td>and</td>
</tr>
<tr>
<td>➢ Think obstruction</td>
<td>➢ Proton pump inhibitors</td>
<td>Ensure clinical context for repeat bloods handed over to those reviewing results</td>
<td>Consider repeating bloods:</td>
</tr>
</tbody>
</table>
Contact Think Kidneys

How to find out more

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