Acute Kidney Injury

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Outline

• What is AKI, and why do we not call it ARF any more?
• AKI – a window on physiological instability
• AKI outcomes and processes in secondary care
• Why is AKI relevant to Primary Care?
  – Prevention
  – Timely identification
  – Management: local guidelines
RIFLE Classification

- **Risk**
  - Increased SCreat x1.5 or GFR decrease > 25%

- **Injury**
  - Increased SCreat x2 or GFR decrease > 50%

- **Failure**
  - Increase SCreat x3
    - OR SCreat ≥4mg/dl
    - Acute rise ≥0.5mg/dl
  - GFR decrease ≥75%

- **Loss**
  - Persistent ARF** = complete loss of kidney function > 4 weeks

- **ESKD**
  - End Stage Kidney Disease (> 3 months)

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**GFR Criteria**

- Urine Output Criteria
  - UO < .5ml/kg/h x 6 hr
  - UO < .5ml/kg/h x 12 hr
  - UO < .3ml/kg/h x 24 hr or Anuria x 12 hrs

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**High Sensitivity**

**High Specificity**
Diagnostic Criteria for Acute Kidney Injury

**Table 2 | Staging of AKI**

<table>
<thead>
<tr>
<th>Stage</th>
<th>Serum creatinine</th>
<th>Urine output</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1.5–1.9 times baseline OR ≥0.3 mg/dl (≥26.5 μmol/l) increase</td>
<td>&lt;0.5 ml/kg/h for 6–12 hours</td>
</tr>
<tr>
<td>2</td>
<td>2.0–2.9 times baseline</td>
<td>&lt;0.5 ml/kg/h for ≥12 hours</td>
</tr>
<tr>
<td>3</td>
<td>3.0 times baseline OR Increase in serum creatinine to ≥4.0 mg/dl (≥353.6 μmol/l) OR Initiation of renal replacement therapy</td>
<td>&lt;0.3 ml/kg/h for ≥24 hours OR Anuria for ≥12 hours</td>
</tr>
</tbody>
</table>
### Acute Kidney Injury, Mortality, Length of Stay, and Costs in Hospitalized Patients

Glenn M. Chertow, Elisabeth Burdick, Melissa Honour, Joseph V. Bonventre, and David W. Bates

- 19,982 patients Boston
- 9205 >1 SCr result

<table>
<thead>
<tr>
<th>Rise in Creatinine Mg/dl (umol/l)</th>
<th>Multivariate OR Hospital mortality</th>
<th>Increase in L.O.S /days</th>
<th>Mean increase Cost ($)</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥ 0.3 (26.4)</td>
<td>4.1</td>
<td></td>
<td>4886</td>
</tr>
<tr>
<td>≥ 0.5 (45)</td>
<td>6.5</td>
<td>3.5</td>
<td>7499</td>
</tr>
<tr>
<td>≥ 1.0 (90)</td>
<td>9.7</td>
<td>5.4</td>
<td>13200</td>
</tr>
<tr>
<td>≥ 2.0 (180)</td>
<td>16.4</td>
<td>7.9</td>
<td>22023</td>
</tr>
</tbody>
</table>
**Figure 3: Evolution of acute kidney injury**

Injury begins before excretory function is lost (i.e., decreased GFR) and can in some cases be detected by the measurements of biomarkers. Such biomarkers can also be used for diagnostic and prognostic assessment. GFR = glomerular filtration rate. NGAL = neutrophil gelatinase-associated lipocalin. Cys C = cystatin C. KIM-1 = kidney injury molecule 1. IL-18 = interleukin 18. GST = glutathione-S-transferase. L-FABP = liver fatty-acid-binding protein. CRP = C reactive protein. IL-6 = interleukin 6.
Pathophysiology

Distinct Entities?
The oxygen sensor, and vulnerability to hypoxic injury
A continuum of diminishing volume sensitivity

Early physiological restoration and avoidance of further ‘hits’ prevents progression of damage and adverse outcomes.

There remains no specific drug therapy for AKI.
AKI – a window on physiological instability

• AKI causes avoidable mortality

Or

• Severe or sub-optimally managed.............causes avoidable mortality

Insert condition that interests you
• AKI – 13-18% hosp admissions

• Mortality 25-30%

• Therefore modest improvement in outcomes will save many lives
Adding Insult to Injury

A review of the care of patients who died in hospital with a primary diagnosis of acute kidney injury (acute renal failure).
Key findings

- 33% of patients had inadequate investigations
- 29% had inadequacies in clinical management
- Poor recognition of acute illness, hypovolaemia and sepsis
Failings in generic care....

- An 85 year old man with significant cardiorespiratory comorbidities and a history of bladder cancer was admitted with abdominal pain under the Surgeons (AKI risk factors not identified).
- He underwent a diagnostic CT scan revealing new left sided hydronephrosis and a femoral hernia causing small bowel obstruction. A laparotomy was performed (AKI risk factors not assessed, and drugs not reviewed accordingly).
- In the 72 hours postoperatively his blood pressure dropped to 90-100 systolic (physiological deterioration not recognised, and therefore not acted upon).
- He continued to receive nephrotoxic and antihypertensive medication as well as renally excreted opiates resulting in coma and aspiration (medication not reviewed).
- His AKI was not recognised despite two AKI alerts being issued on Sigma (current alert system, and response to alerts, insufficiently robust).
- There was no plan for decompression of his obstructed urinary tract (delays in recognition and management of urinary tract obstruction).
- He was anuric for 24 hours prior to renal referral by which point he was critically unwell and required urgent dialysis and nephrostomy insertion (poor fluid balance recording, resuscitation and delayed referral).
Acute Kidney Injury (AKI) Programme

Acute Kidney Injury (AKI) is an emerging global healthcare issue. As healthcare increases in complexity, the interaction between long term medical conditions, medication and inter-current illness are too often complicated by AKI. It is estimated that one in five emergency admissions into hospital are associated with AKI (Wang et al, 2012), that up to 100,000 deaths in secondary care are associated with AKI and that 1/4 to 1/3 have the potential to be prevented (National Confidential Enquiry into Patient Outcome and Death (NCEPOD) Adding Insult to Injury 2009).

The resource and economic burden upon the healthcare economy is considerable. It is estimated that the additional cost is £500 milion (data from NHS Kidney Care 2012).
Acute kidney injury: Prevention, detection and management of acute kidney injury up to the point of renal replacement therapy

New NICE kidney guideline to save thousands of lives

The National Institute for Health and Care Excellence (NICE) has published a new guideline which promises to save thousands of lives and hundreds of millions of pounds each year. The new guideline will help prevent, detect and treat acute kidney injury (AKI), a condition that affects one in six people who are admitted to hospital and although it is completely preventable, can lead to death in one in four of those.

Evidence suggests a lack of education about the condition among healthcare workers. The NICE guideline aims to raise awareness and recommends that AKI is tackled by people working in health across all specialties, not just renal units, from chief executives to healthcare assistants.
Ensuring timely detection of AKI: plan for roll-out to primary care

Patient Safety Alert

Stage Three: Directive
Standardising the early identification of Acute Kidney Injury
9 June 2014

Who: NHS acute trusts and foundation trusts providing pathology services
When: By 9 March 2015

Alert reference number: NHS/PSAVD/2014/010
Alert stage: Three - Directive

National patient safety data tells us that patients are dying and suffering severe harm due to a delay in detecting Acute Kidney Injury (AKI). AKI often occurs without causing any symptoms or signs and its presence frequently goes unrecognised by patients and doctors alike.

“A patient with a complex physical and mental health background became unwell over a weekend. Despite persistent hypotension there was no record of fluid balance. Bloods were delayed until late Sunday night, indicating acute kidney injury. Acute kidney injury not recognised or commented on until mid way through the following day. Medications given to the patient over the weekend included drugs contraindicated in renal failure. The patient was admitted to ICU and on admission was unconscious/shocked. There were multiple systematic failures in the management of this patient including a life threatening delay in critical care of >12 hours and systems failure in the recognition of deteriorating patients.”

Although primary care is an important focus for detection and prevention of AKI, it is anticipated that AKI results will be sent to primary care in a second phase of the programme. Meanwhile Trusts are expected to discuss with primary care representatives the management of AKI test results, particularly at times when deputizing services are providing medical cover.
‘Community acquired’ AKI accounts for two-thirds of cases

Selby NM et al CJASN 2012; 7(4): 533
Service improvement must occur across organisational boundaries.
Editorials
Acute kidney injury in the community:
why primary care has an important role

• Recognition of patients at risk of AKI – medicines management and ‘sick day rules’
• Importance of timely treatment of sepsis and fluid balance
• Follow up: risk of CKD
• Raised creatinine = AKI until proved otherwise

REASONS FOR FOCUSING ON ACUTE KIDNEY INJURY
There is mounting evidence that awareness of kidney function is central to the delivery of safe and clinically-effective care, in terms of preventing both cardiovascular events, and progression to established renal failure, with significant impacts on quality of life and healthcare expenditure. However,

“...UK general practice is in a unique position to identify people at increased susceptibility to AKI and address potentially modifiable exposures.”
Community Acute Kidney Injury (AKI) Guideline

AKI alert received from laboratory
Check patient’s previous urea, creatinine and electrolyte results [U&E’s] to differentiate from stable Chronic Kidney Disease [CKD]
OR
AKI diagnosed on serial U&E results

☐ Review by GP / community nurse practitioner
☐ Diagnose & treat any acute illnesses contributing to AKI

*Essentials steps to be initiated by community physicians/ team*

*think FLUIDS:*

☐ **Fluid balance:** Check for signs of dehydration and treat. (Encourage oral fluid)
☐ **Low BP** (check BP and if low, SBP<110), withhold anti-hypertensive, diuretics (If history of angina/ cardiac arrhythmia, reduce beta blocker)
☐ **Urine:** dip test and microscopy. (Look for signs of urinary retention)
☐ **Bladder palpable:** Catheterise
☐ **Drugs and Toxins:** Stop NSAID, COX 2 Inhibitors, Trimethoprim. ACE Inhibitors, Angiotensin receptor blockers. Avoid any nephrotoxic medications
☐ **Sepsis:** Look for signs of sepsis and treat accordingly
Who to admit

If Se K is > 5.7 mmol/L, refer in to AED, unless advance directive states otherwise

If patient unwell and not responding clinically, or remains oliguric, or requires IV therapy, refer in to AED unless advance directive states otherwise

If patient is well,

- Discuss with Renal on-call team or Acute Medicine [Medical Admissions Unit/ Emergency Admission Unit]
- Repeat U & E’s in 24 - 48 hours
- Renal screen*: if haematuria/proteinuria or, suspected vasculitis/ myeloma
- Continue with essential steps listed above
- Refer to hospital if vasculitis or acute nephritis suspected/ despite treatment worsening U&E’s or persistent oliguria, unless advance directive states otherwise