Acute Kidney Injury (AKI)
Undergraduate nurse education
Year Three

Developed Summer 2017
Objectives

- Understand Acute Kidney Injury and its relevance to patient care.
- Brief revision of the Anatomy and physiology of the kidneys
- Establish the aetiology, risk factors and staging of AKI
- Understand the investigations and tests that will aid diagnosis
- Recognise the role of the MDT in AKI
- Understand your role in the early recognition and management of AKI
What is Acute Kidney Injury (AKI)?

AKI is the universal term used to describe sudden deterioration of renal function, replacing Acute Renal Failure. It is a spectrum of injury which if unrecognised can lead to renal failure and death (damage → failure → death). It’s seen in the community and on all hospital wards.

Renal function will continue to deteriorate unless AKI is recognised and its cause identified and treated.
## Identifying AKI

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<th>Urine Output</th>
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<td>$&lt;0.5 , ml/kg/hr$ for $&gt;8$ hrs</td>
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<td>2</td>
<td>Increase to $&gt;200$-300% of baseline ($&gt;2$-3 fold)</td>
<td>$&lt;0.5 , ml/kg/hr$ for $&gt;12$ hrs</td>
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<td>Increase to $&gt;300%$ of baseline ($&gt;3$ fold) or serum creatinine greater than $&gt;354 , \mu{mol/l}$ (4mg/dl) with an acute rise of at least $44 , \mu{mol/l}$ (0.5mg/dl)</td>
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- AKI is detected by a rise in Creatinine (Cr) and/or a decrease in urine output
- Its severity (stage) is related to the change from the patient’s baseline Cr
- It can occur without symptoms
This graph shows that regardless of stage survival at 1 year post AKI was only 50-60%.

Consider the small change in Creatinine/urine output needed to trigger a stage 1 AKI.
The UK National Confidential Enquiry into Patient Outcome and Death (NCEPOD) report ‘Adding insult to injury’ http://www.ncepod.org.uk/2009aki.html found:

- Only 50% of care for AKI was considered good
- Unacceptable delays in identifying post admission AKI in 43% of the patients
- Considerable knowledge gaps in identifying and managing someone with AKI
- A fifth of all cases were predictable and avoidable

**Predictable, avoidable AKI should not occur**
The NHS campaign to improve the care of people at risk of, or with, acute kidney injury

In the UK up to 100,000 deaths each year in hospital are associated with acute kidney injury. Up to 30% could be prevented with the right care and treatment. NCEPOD, Adding Insult to Injury, 2009

One in five people admitted to hospital in the UK each year as an emergency has acute kidney injury. Wang, et al. 2012

Just one in two people know their kidneys make urine. Ipsos MORI survey, July 2014

About 65% of acute kidney injury starts in the community. Selby, et al. 2012

Think Kidneys is a national programme led by NHS England in partnership with UK Renal Registry.

Acute Kidney Injury Year Three
Anatomy and Physiology recap
What do the Kidneys do?

1. remove waste products and drugs from the body
2. regulate the water content of the body
3. regulate electrolytes in the blood
4. regulate acid-base balance
5. release hormones that regulate blood pressure
6. produce an active form of vitamin D that promotes strong, healthy bones
7. control the production of red blood cells
To function kidneys need...

- Adequate renal blood flow and oxygenation
- To be healthy, rather than damaged or inflamed by disease processes
- To drain urine freely through a functioning urinary tract
Who is at risk and how is it caused?
Risk factors

- chronic kidney disease
- heart failure
- liver disease
- diabetes
- history of acute kidney injury
- oliguria (urine output less than 0.5 ml/kg/hour)
- Hypovolaemia
- Sepsis
- deteriorating early warning scores

Drugs can also affect kidney function, these include:

- NSAIDs,
- angiotensin-converting enzyme [ACE] inhibitors,
- angiotensin II receptor antagonists [ARBs] and diuretics within the past week, especially if hypovolaemic
- use of iodinated contrast agents
- symptoms or history of urological obstruction, or conditions that may lead to obstruction
- Age 65 years or over

Are any of your patients at risk??
Pre- Renal
Most common cause of AKI and caused by reduced blood flow to the kidneys

Causes include:
• Hypotension
• Dehydration
• Heart Failure

Assess hydration and NEWS

With prompt correction this AKI rapidly resolves

Intrinsic
Involves damage to the kidney itself. Some causes include:
• Acute tubular necrosis
• Glomerulonephritis
• Drugs/toxins

Hardest to treat and likely to need Renal Team involvement

A urine dip will aid diagnosis

All AKI will eventually become intrinsic if it is not treated promptly

Post-Renal
A consequence of urinary tract obstruction

• Blocked catheter
• Enlarged prostate
• Tumours

An obstruction can be above or below the bladder so consider bladder scan +/- USS KUB

May need a Urology review

Treatment of AKI depends on its cause
Pre-renal factors in preventing AKI-HYDRATION

Assessing hydration is essential for the prevention and management of AKI.

Dehydration is the most common cause of pre-renal AKI.

Intravenous fluid are often used to treat pre renal AKI. Only 72% of 366 patients seen by an AKI Team in a small scale study actually received them as prescribed.

In a hydrated patient with stable NEWS, rule out obstruction and consider intrinsic causes!

Healthy wee is 1-3, 4-8 you must hydrate!

REMEMBER
Fluid Balance
Urine Colour
Daily weights
Lying/standard BP
Why is fluid balance so important?

Accurate fluid balance charting can also prevent life threatening complications of AKI
All patients with AKI should have a urine dip. **Blood** and **Protein** in urine is a sign of kidney disease and intrinsic AKI.

**THINK:**

Have your patients passed urine in the last 6 hours?
Cumulative totals should be done every 6 hours as well as daily.
Is your fluid balance chart accurate? In a small scale study in one trust, 85% of 570 patients seen by the AKI team did NOT have accurate in/out put monitoring!
What is an expected urine output of a patient?

Expected urine output is 0.5mls/kg/hr. If less than this, escalate!

Chris Boateng is a 65 year old lady who has come in for a knee replacement. She weighs 90kg. How much urine should she pass in an hour?

• In the last 6 hours Chris has passed 300 mls of Urine. Is this a concern?

• Chris was kept in overnight due to complications. Her fluid balance has a total of 800mls of urine passed since admission. What do you do?
Why review medications?

Some medications can impair renal function? Can you think of any that you have come across in your training?

- Contrast media
- ACE Inhibitor/ Angiotensin receptor blocker
- NSAIDs
- Diuretics

How do these effect your kidneys/ kidney function?
Why review medications?

Some medications are excreted via the kidneys and have the potential to accumulate during AKI

- Do you know any drugs that can accumulate in AKI?
- What side effects can these cause?
- How can you reduce the risk of accumulation?

All patients with AKI must have a medication and pharmacist review
Plan of management for a patient with AKI
Confirming AKI and identifying potential cause

In response to the findings of the NCEPOD (2009) report NICE published AKI Guidelines in 2013 (CG 169). Trusts have used these to develop their own AKI Policies and bundles.

You are told that your patient has an AKI/ you notice that they have a reduced urine output. What do you do next?

- Does your Trust have an AKI policy or bundle?
- Reflect on what these recommend and why
The London AKI network has lots of useful resources consider
Sepsis and hypoperfusion
Toxicity
Obstruction
Primary renal disease
http://www.londonaki.net/clinical/guidelines-pathways.html
ACUTE KIDNEY INJURY PATHWAY

**AKI Stage 1**
- ↑ Cr > 26μmol/l past 48 hr
- OR
- ↑ Cr to 150-200% (1.5-2 x baseline)
- OR
- Urine output <0.5ml/kg/hr > 6hrs

**AKI Stage 2**
- ↑ Cr to 200-300% (2-3 x baseline)
- OR
- Urine output <0.5ml/kg/hr > 12 hrs

**AKI Stage 3**
- ↑ Cr to >300% (>3 x baseline)
- OR
- Urine output <0.3ml/kg/hr > 12 hrs

**AKI**

1. Complete set observations (with postural BP if able)
2. Alert medical staff and request senior review (ST3 or above)
3. NEWS score. Consider CCOT input
4. ECG, consider monitor
5. IV access, bloods (U&E, FBC, LFT, HCO₃, Ca, PO₂, CRP, venous blood gas-lactate)
6. Urinalysis
7. Assess for sepsis
8. Review medications, stop ALL nephrotoxic agents, dose adjust all prescribed medications for GFR

**BP systolic <90mmHg AND no evidence fluid overload** GIVE 250-500ml fluid bolus

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If hyperkalemic ECG changes GIVE 10ml 10% calcium chloride
Potassium > 6.5mmol/L GIVE 10 units short acting insulin with 50ml 50% glucose, monitor blood glucose 1hrly
Bicarbonate < 20mmol/L - consider referral to renal

REVIEW PATIENT
BP MAP < 65mmHg OR urine output <0.5ml/kg/hr- ECOT review
IF CLINICALLY APPROPRIATE URGENT RENAL AND ITU REFERRAL

AKI stage 1/ stage 2
Minimum 4 hourly observations, 1 hourly if triggering
Monitor urine output, consider catheter
Daily weights and fluid balance
Daily U&E
Assess need to continue fluids
Consider need for renal tract ultrasound
Continued clinical review/ out of hours handover
AKI stage 2 - consider renal referral

AKI stage 3
1 hourly observations
Catheterise and record hourly urine output
Repeat bloods with venous gas within 4-6hrs or arterial gas if respiratory compromise
Daily weights and fluid balance
Daily U&E and arrange ultrasound renal tract
Refer RENAL and handover to medical registrar on-call

Case study
Patient David Smith

Situation

68 year old gentleman admitted to Emergency Department with collapse

Background

Patient states feeling unwell for last 5 days and hasn’t been eating and drinking as much as usual

Past medical history

Chronic diabetic foot ulcer

Hypertension

Ischemic Heart Disease

NSTEMI

Type 2 diabetes

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<tr>
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<th>Dose</th>
<th>Timing</th>
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<td>40mgs</td>
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Assessment

A. Clear

B. RR 19, SpO2 95% on room air, no Shortness of Breath (SOB)/difficulty in breathing

C. BP 86/50, HR 94, Temp 38.2, Capillary refill time: 3 secs, cool peripheries, dry mucus membranes

D. Alert, Capillary Blood Glucose: 6.7, no pain

E. No peripheral oedema, bilateral leg ulcers

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<td>Baseline</td>
</tr>
<tr>
<td>On admission</td>
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Does he have an AKI and if so what stage?

Take a few minutes to decide and feed back to the class:

- Does he have an AKI and if so what stage?
- What are his risk factors for AKI?
- What would be your recommendations be?

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Assessment

David has stage 2 AKI

- To work out what stage he is:
  - \( \text{admission creatinine} \div \text{baseline creatinine} = \)
  - \( \frac{145}{70} = 2.1 \)

Risk Factors

His risk factors for AKI

- Age
- Diabetes
- Heart disease
- Drugs?
- Diuretics
- Hypotension
- Sepsis

What would be your recommendations?
Recommendations

- NEWS monitoring
- ECG
- Bloods: (urea and electrolytes, Liver function tests (LFT’s), Full blood count (FBC) venous lactate
- Urinalysis - what are you looking for?
- Assess and correct dehydration; IVF, Strict fluid balance, lying and standing BP
- Rule out sepsis
- Escalate to medical team (ST3 or above)
- Review medications
Urine output

David reports that he has been passing urine but that it has been less frequently than normal and its appearance is much darker

- What could this mean?
- How can we measure David’s urine output?
- Does he need a catheter?

If he weighs 85kgs

- What should his urine output be in mls/hr?
Can any of his medications affect his renal function?

**Furosemide** and **spironolactone**: these are both diuretics. A decrease in fluid intake could cause dehydration.

**Ramipril** is an **ACE inhibitor**. These are antihypertensives and have nephrotoxic potential when a patient is dehydrated. Also associated with hyperkalaemia.

**Metformin** is associated with lactic acidosis in AKI and can accumulate causing hypoglycaemia.

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His medications were reviewed and with fluid resuscitation his BP improved and AKI resolved. His NEWS score was stable at 0 and his usual medications were restarted.

**However**

... David’s CRP continued to increase so he was given 3 doses of Gentamicin and a CT with IV contrast to look for source of sepsis...

What are his risk factors for AKI now?
Additional risk factors: Gentamicin, IV contrast and previous AKI

**How could we manage this risk? What could you do to prevent another AKI?**

- Regular observations
- Optimise hydration
- Medication review
- Fluid balance monitoring
- Daily U+E’s

Despite this...
<table>
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</tr>
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<td>Day 1</td>
<td>145</td>
</tr>
<tr>
<td>Day 2</td>
<td>152</td>
</tr>
<tr>
<td>Day 5</td>
<td>222</td>
</tr>
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What stage AKI is this and what would you recommend now?
Care as per AKI guidance

- NEWS monitoring
- ECG
- Bloods: (urea and electrolytes, Liver function tests (LFT’s), Full blood count (FBC) venous lactate
- Urinalysis - what are you looking for?
- Assess and correct dehydration; IVF, Strict fluid balance, lying and standing BP
- Rule out sepsis
- Escalate to medical team (ST3 or above)
- Review medications
Strict Fluid Balance is required in order to identify whether the kidneys are producing enough urine. Positive or negative balance could help identify dehydration or risk of fluid overload. Giving fluid to someone who is overloaded with fluid could result in pulmonary oedema and heart failure.

If David weighs 85Kg is he passing enough urine?
Further information

NEWS=0, CVS stable
Pt alert, eating and drinking and well hydrated
Medications reviewed and withheld as before
Urine dip:

<table>
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<tr>
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<tr>
<td>Nitrites</td>
<td>Negative</td>
</tr>
<tr>
<td>Leucocytes</td>
<td>Negative</td>
</tr>
<tr>
<td>Protein</td>
<td>Positive</td>
</tr>
<tr>
<td>Blood</td>
<td>Positive</td>
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</table>

What does this urine dip suggest?
What can cause these results?

What other investigations should be considered and why?
Further investigations

- **US KUB** (Ultrasound of kidneys, ureters and bladder)

  No abnormalities detected, no signs of obstruction

- **VBG** (Venous blood gas)

<table>
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<th>Value</th>
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<tr>
<td>Ph</td>
<td>7.30</td>
</tr>
<tr>
<td>PC02</td>
<td>6.52</td>
</tr>
<tr>
<td>Bicarbonate</td>
<td>18</td>
</tr>
<tr>
<td>Urea</td>
<td>26</td>
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What is the likely cause of David’s AKI?
Outcome

This time his AKI was intrinsic and caused by a problem within the kidneys themselves.

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</tr>
<tr>
<td>Day 6</td>
<td>476</td>
</tr>
<tr>
<td>Day 7</td>
<td>520</td>
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David was transferred to renal ward and was considered for dialysis. Once his renal function stabilised he had a right femoral endarterectomy and right foot debridement. He stayed in hospital for 31 days.
Referral to Renal Team

Not all patients with an AKI need a Renal review (NICE 2013). Is there a clear cause and is the patient responding to medical management?

Discuss with renal when there is:

- a possible diagnosis that may need specialist treatment (for example, vasculitis, glomerulonephritis, tubulointerstitial nephritis or myeloma)
- acute kidney injury with no clear cause
- inadequate response to treatment
- stage 3 acute kidney injury
- a renal transplant
- chronic kidney disease stage 4 or 5
- complications associated with acute kidney injury
Indications for Renal Replacement Therapy

A patient may require renal replacement therapy in the form of Haemodialysis or Haemofiltration if the patient does not respond to medical management for:

- hyperkalaemia
- metabolic acidosis
- symptoms or complications of uraemia (for example, pericarditis or encephalopathy)
- fluid overload
- pulmonary oedema.

Acute Kidney Injury Year Three
Any questions?