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

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



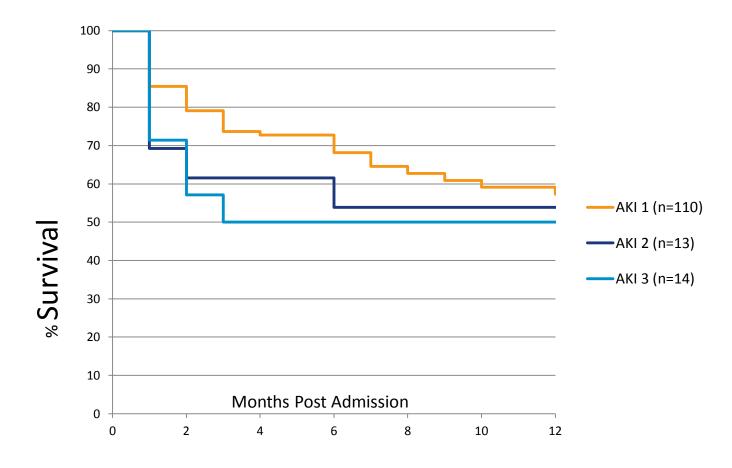
Identifying AKI

Stage	Serum Creatinine	Urine Output
1	Increase of ≥ 26.4 µmol/I (0.3mg/dl) OR to 150-200% of baseline (1.5-2.0 fold)	<0.5 ml/kg/hr for >6hrs
2	Increase to >200-300% of baseline (>2-3 fold)	<0.5 ml/kg/hr for >12hrs
3a	Increase to >300% of baseline (>3 fold) or serum creatinine greater than <u>></u> 354 µmol/l (4mg/dl) with an acute rise of at least 44 µmol/l (0.5mg/dl)	<0.3ml/kg/hr for 24hrs OR anuria for 12 hrs

- AKI is detected by a rise in Creatinine (Cr) and/or a decrease in urine output
- It's severity (stage) is related to the change from the patients baseline Cr
- It can occur without symptoms



In-Hospital AKI from March-April 2014



Consider the small change in Creatinine/ urine output needed to trigger a stage 1 AKI

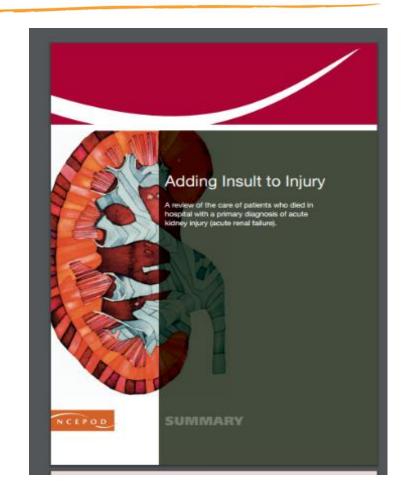
This graph shows that regardless of stage survival at 1 year post AKI was only 50-60%.

Acute Kidney Injury (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

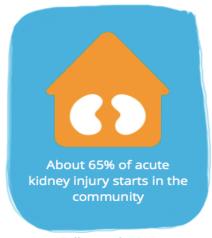
NCEPOD. Adding insult to injury, 2009



Wang, et al. 2012



Ipsos MORI survey, July 2014



Selby, et al. 2012







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

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
- (NICE Guideline 169, 2014)

- Orugs can also affect kidney function, these include:
 - SAIDs,
 - 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



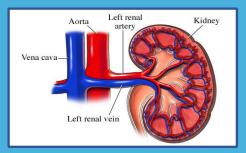
Are any of your patients at risk??

Treatment of AKI depends on its cause



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

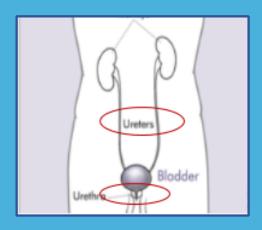
Hardest to treat and likely to need Renal Team involvement

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

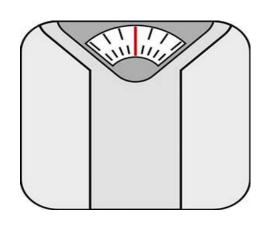
An obstruction can be above or below the bladder so consider bladder scan +/- USS KUB

May need a Urology review

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

REMEMBER

Fluid Balance
Urine Colour
Daily weights
Lying/standard BP

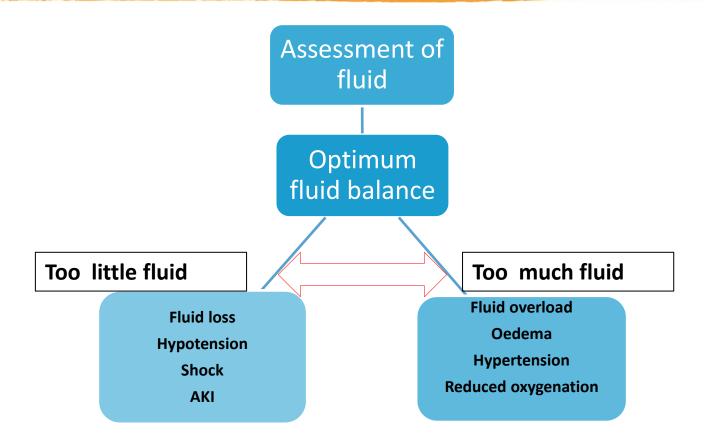


Healthy wee is 1-3, 4-8 you must hydrate!

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



Why is fluid balance so important?



Accurate fluid balance charting can also prevent life threatening complications of AKI



DIP, MEASURE & DOCUMENT

			Urinary	Analysis			
Date	рН	Blood	Protein	Glucose	Ketones	Nitrites	Leucocyte
			15-12/10/H-F-1-X-				
	1					3	9
						70	

Daily Weight		Blood
Date	Weight	Pressure

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!





Any care concerns, document on the relevant shee



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?

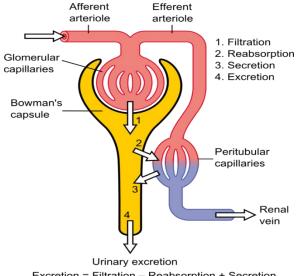


Why review medications?

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?



Excretion = Filtration - Reabsorption + Secretion



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

Diagnose the Cause

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

This is a Medical Emergency

Full set of observations, circulatory assessment, treat life-threatening complications, if NEWS triggering give oxygen, begin resuscitation and contact critical care outreach team

Diagnose the cause(s) and treat all – STOP AKI
Sepsis and hypoperfusion, Toxicity, Obstruction, Primary renal disease

Sepsis and hypoperfusion

Circulatory assessment
(history, heart rate,
blood pressure, JVP,
capillary refill (should be
<3 secs), conscious level
Bolus fluids (e.g. 250-500mls
balanced crystalloid) until
volume replete with regular
review of response.

Senior review if no response 2 litres filling Stop anithypertensives if relative hypotension Infection/sepsis screening (history, examination, cultures, CRP) and antibiotics if suspected If severe sepsis 'sepsis six'

and antibiotics < 1 hour

Toxicity

Ascertain full drug history including contrast exposures Avoid further nephrotoxic insults if possible Stop ACE/ARB

Stop NSAID

If poisoning AKI (e.g. lithium, ethylene glycol) get specialist renal and toxicology help

Obstruction

Ascertain any urological history. High index of suspicion if malignancy

Examine or bedside scan for bladder, consider urinary catheter.

Perform renal tract imaging (ultrasound or CT KUB) <24 hours unless non-obstructive cause clear.

If obstructed and infected urinary tract suspected (pyonephrosis) imaging <6 hours.

If likely/suspected obstructed AKI refer urology.

Target time to relief of obstruction 12 hours after diagnosis, immediate if infected.

Primary renal disease

Ascertain relevant history (e.g. autoimmune disease, myeloma, HUS/TTP)

Urine dipstick (all AKI patients). If protein high measure PCR.

Check CK (rhabdo), CRP, FBC, If platelets low do blood film, bill, LDH, relics (HUS/TTP)

Consider myeloma screen (Igs, Ig electrophoresis, serum free light chains, urine bench jones

Consider renal immune screen (ANCA, anti-GBM, ANA, complement, rheumatoid factor, Igs)

If likely/suspected primary renal injury refer nephrology

General supportive care and escalation

Once euvolaemic give maintenance fluids (e.g. output plus 500mls),
fluid chart, daily weights, regular fluid assessment
Regular (at least 4 hourly) observations/NEWS with clear escalation plans
Review all drug dosages, consider proton pump inhibitor, consider dietetic review and nutrition
Urea, electrolytes, bone and venous bicarbonate at least daily, consider ABG
Monitor for complications, treat and escalate

Severe AKI (AKI 3) should be discussed nephrology and critical care regardless of cause



Current Cr

Baseline Cr (lowest in past 3 mths)

East and North Hertfordshire NHS NHS Trust

ACUTE KIDNEY INJURY PATHWAY

AKI Stage 1

† Cr > 26umol/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 >12hrs

AKI

- Complete set observations (with postural BP if able)
- Alert medical staff and request senior review (ST3 or above).
- **NEWS** score. Consider CCOT input
- ECG, consider monitor
- IV access, bloods (U&E, FBC, LFT, HCO₃, Ca, PO₄ CRP venous blood gas-lactate)
- Urinalysis.
- Assess for sepsis
- 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



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- CCOT 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

www.londonaki.net

www.nice.org.uk

www.renal.org

www.akinet.org

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

Medications		
Furosemide	40mgs	OM
Spironolactone Ramipril	50mgs 10mgs	OM OM
Aspirin	75mgs	ОМ
Atorvastatin	80mgs	ON
Metformin	500mgs	BD



Assessment

	Creatinine
Baseline	70
On admission	145

- 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

Does he have an AKI and if so what stage?

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

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

Stage	Serum Creatinine	Urine Output
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Assessment

David has stage 2 AKI

- To work out what stage he is:
- **admission creatinine** + baseline creatinine=

$$145 \div 70 = 2.1$$

Risk Factors

His risk factors for AKI

- Age
- diabetes
- heart disease
- Orugs?
- 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?
- Ooes he need a catheter?

If he weighs 85kgs

What should his urine output be in mls/hr?



Medication Review

Medications		
Furosemide	40mgs	OM
Spironolactone Ramipril	50mgs 10mgs	OM OM
Aspirin	75mgs	ОМ
Atorvastatin	80mgs	ON
Metformin	500mgs	BD

Can any of his medications affect his renal function?

Frusemide 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.



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...



Days in hospital	Creatinine
Baseline	70
Day 1	145
Day 2	152
Day 5	222

What stage AKI is this and what would you recommend now?

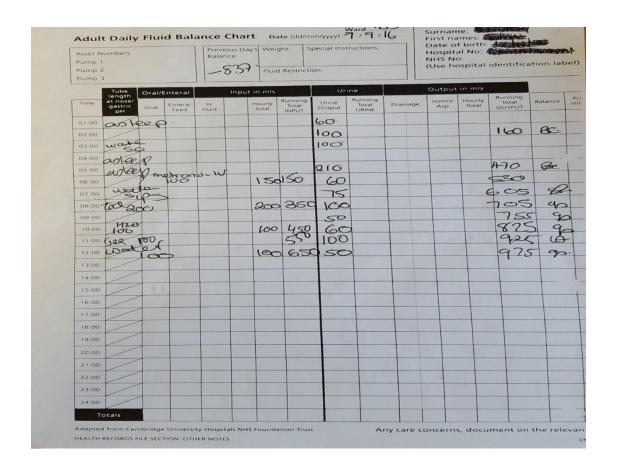


Care as per AKI guidance

- 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
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Fluid Balance chart



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:

Urine dip	
Nitrites	Negative
Leucocytes	Negative
Protein	Positive
Blood	Positive

What does this urine dip suggest?

What can cause these results?

What other investigations should be considered and why?



Further investigations

• <u>US KUB</u> (Ultrasound of kidneys, ureters and bladder)

No abnormalities detected, no signs of obstruction

VBG (Venous blood gas)

VBG	
Ph	7.30
PC02	6.52
Bicarbonate	18
Urea	26

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

Days in hospital	Creatinine
Baseline	70
Day 1	145
Day 2	152
Day 5	222
Day 6	476
Day 7	520

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
- **c** 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.



Any questions?

