Preventing Acute Kidney Injury

Dr Andy Lewington MSc Clin Ed FRCP
Consultant Nephrologist/Honorary Clinical Associate Professor
Director of Undergraduate Medical Education
Leeds Teaching Hospitals
A.J.P.Lewington@leeds.ac.uk
Leeds Teaching Hospitals/University of Leeds
Declaration of Interest

- AM Pharma – Advisory Board and Co Chief Investigator for Alkaline Phosphatase therapy for AKI
- Bioporto – Advisory Board for NGAL
- Fresenius – Honoraria for lecturing and support for Leeds Critical Care Nephrology Conference
- Baxter – Honoraria for lecturing on IV Fluids
Outline

• Describe the syndrome of AKI
• Highlight the importance of preventing this medical emergency and preserving kidney function acutely and long term
• Consider which patients are at risk of AKI
• Propose a STOP AKI management plan
'The patient must be the first priority in all of what the NHS does. Within available resources they must receive effective services from caring compassionate and committed staff working within a common culture, and they must be protected from avoidable harm and any deprivation of their basic rights.'

(Robert Francis QC)\(^1\)

'Patient safety should be the ever-present concern of every person working in or affecting NHS-funded care. The quality of patient care should come before all other considerations in leadership and conduct of the NHS, and patient safety is the keystone dimension of quality.'

(Don Berwick, 2013)\(^2\)
Patient Safety Agenda - ‘zero harm’
Acute kidney Injury – A marker of Quality?

• ‘AKI as the single measure which will tell us if we are making progress...’

• ‘If we can get it right for AKI, we will get basic care right across the NHS.’

Professor Donal O’Donoghue
Former National Clinical Director for Kidney Care
BBC, 2013
What is Acute Kidney Injury?
AKI is a Syndrome

**Intrinsic AKI**
- Acute tubular injury
  - Prolonged pre-renal AKI
  - Rhabdomyolysis
  - Haemoglobinuria
  - Nephrotoxins
    - Iodinated contrast
    - NSAIDs
    - Gentamicin
- Tubulointerstitial injury
- Glomerulonephritis
- Myeloma
- Vasculitis
  - Lupus
  - ANCA associated

**Pre-renal AKI**
- Sepsis
- Hypovolemia
  - Haemorrhage
  - Burns
  - Vomiting/Diarrhoea
  - Diuretics
- Hepato-renal Syndrome
- Cardiac Failure
- Hypotension
  - Medications

**Post-renal AKI**
- Kidney stones
- Prostatic hypertrophy
- Tumours
- Retroperitoneal fibrosis
Acute Kidney Injury

- Most commonly associated with acute illness
  - Sepsis
  - Hypotension
- Life threatening complications include
  - Hyperkalaemia
  - Acidaemia
  - Pulmonary oedema
  - Pericarditis
  - Encephalopathy
Acute Kidney Injury

• Outcomes
  – ↑ mortality
  – ↑ length of stay
  – chronic kidney disease (cardiovascular risk)

• Cost

• A degree of recovery usual if patient recovers from primary cause
Acute Kidney Injury

• Rarer forms
  – require rapid recognition for specific therapy
    • e.g. vasculitis – Lupus/ANCA associated
  – systemic symptoms
    • fever
    • failure to thrive
  – rash
  – joint pains
  – active urinary sediment – blood and protein
  – **CHECK the kidney function early**
The Risk of Dying From AKI is Higher than Dying from
Myocardial Infarction
or
Breast cancer,
Prostate cancer, Heart failure
and Diabetes
combined
Increased mortality associated with changes in serum creatinine

Odds of death

- Unadjusted
- Age- and sex-adjusted
- Multivariable-adjusted

Chertow et al: JASN 2005
AKI Definition – KDIGO

<table>
<thead>
<tr>
<th>AKI stage</th>
<th>Serum Creatinine criteria</th>
<th>Urine output criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>SCr increase ≥26 µmol/L within 48 hrs</td>
<td>&lt;0.5 mL/kg/hr for 6 consecutive hrs</td>
</tr>
<tr>
<td></td>
<td>or</td>
<td></td>
</tr>
<tr>
<td></td>
<td>SCr increase 1.5–1.9 fold from baseline</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>SCr increase 2–2.9 fold from baseline</td>
<td>&lt;0.5 mL/kg/hr for 12 hrs</td>
</tr>
<tr>
<td>3</td>
<td>SCr increase ≥3 fold from baseline</td>
<td>&lt;0.3 mL/kg/hr for 24 hr</td>
</tr>
<tr>
<td></td>
<td>or</td>
<td></td>
</tr>
<tr>
<td></td>
<td>SCr increase ≥354 µmol/L</td>
<td></td>
</tr>
<tr>
<td></td>
<td>or</td>
<td></td>
</tr>
<tr>
<td></td>
<td>initiated on RRT (irrespective of stage at time of initiation)</td>
<td></td>
</tr>
</tbody>
</table>
Changing Face of Medicine
Estimated and projected UK population mid-2008 and mid-2033

Source: ONS
Hospital Population

• Many patients have
  – Multiple co-morbidities
  – More complex management issues
  – Decreased functional reserve
    • Cardiac
    • Respiratory
      • Kidney – natural decline in function after 40 years
  – Polypharmacy – e-prescribing

• Education
  – promote an integrated approach
If 20% - 30% of AKI is preventable

- 28,000 – 42,000 preventable cases of AKI in English hospitals each year based on HES – conservative estimates
- 8,000 – 12,000 preventable AKI-related deaths each year
The Economic Impact of AKI in England

• Cost of AKI is 1.1% of the NHS Budget

• AKI costs NHS more than three of the four most common cancers combined

• Costs do not include
  – community acquired AKI or
  – long-term costs other than dialysis

• Prevention of 20% of cases would pay
  – salaries and overheads for 20 to 30 extra nurses at every acute Trust
Clinical Case
Clinical Case

• 76 yrs old Female
• Undergoing major spinal surgery
• PMH
  – Type II Diabetes Mellitus – 6 years
  – Hypertension – 8 years
    • Baseline BP 130/80 mmHg
  – Heart Failure – 3 years
  – Creatinine 85 umol/L
    • (eGFR 55 mls/min/1.73m²)
Clinical Case

• Medications
  – Metformin
  – Enalapril
  – Amlodipine
  – Bisoprolol
  – Gliclizide
  – Omeprazole
  – Spironalactone
  – Furosemide
  – Dihydrocodeine
  – Gabapentin
  – Aspirin

• High Risk Patient
• ? Hold antihypertensives
Box 1 Risk factors for AKI

- Age >75 years
- Pre-existing CKD (eGFR <60 mL/kg/1.73 m²)
- Previous episode of AKI
- Debility and dementia
- Heart failure
- Liver disease
- Diabetes mellitus
- Hypotension (mean arterial pressure <65 mmHg, systolic pressure <90 mmHg)
- Sepsis
- Hypovolaemia
- Nephrotoxins, eg gentamicin, NSAIDs, iodinated contrast
- Antihypertensives in setting of hypotension, eg ACE inhibitors, loop diuretics

ACE = angiotensin-converting enzyme; eGFR = estimated glomerular filtration rate; NSAIDs = non-steroidal anti-inflammatory drugs
Clinical Case

• 5 hour op
  – urinary catheter
  – BP dropped 60-80 mm/Hg
  – Gentamicin
  – Paracoxib
  – *No urine output charted*

• 4 hrs post op returned to HDU

• *Risk of AKI increased*
Clinical Case

- Orthopaedic F2 called me at 12.30am
  - U.O. 136 mls since urinary catheter
  - 5litres of 0.9% sodium chloride
  - Creatinine 158 umol/L
  - K 4.8 mmol/L
  - ‘should we give more fluid?’
Clinical Case

• AJPL
  – Patient was at high risk of AKI
  – ABCDE assessment
    • A – safe
    • B – $O_2$ Sats 98%, RR 16/min, lungs clear, on 2 L nasal cannula
    • C – well perfused, PR 62/min, BP 90/55mmHg, oliguric
    • D – Alert
    • E – T 37.2 $^0$C no evidence of infection
Clinical Case

- **NEWS 4**
  - low risk
    - monitor 4 hrly
    - registered nurse must assess and decide on monitoring and/or escalation

- **Urine Output**
  - 136 mls over 9 hrs
  - *Not part of NEWS*

- **AKI**
  - Stage 1 on Cr criteria
  - Should be part of NEWS???
Clinical Case

– STOP AKI Management Plan

• **Sepsis 6** – low threshold for cultures, CXR

• **Toxins** - ? Gentamicin/NSAID – Avoid further

• **Optimise BP** – Hold Furosemide/Spironalactone/ACE i/Amlodipine, avoid further fluids if pulmonary oedema on CXR

• **Prevent Harm** – hold Metformin, Gabapentin, careful with opiates, check Bicarbonate
  – identify cause
  – review fluid management plan

– Recommend ICU review
Clinical Case

• Following day
  – called by ICU consultant 12pm – told team to stop fluids
  – AJPL reviewed patient 5pm
    • A – safe
    • B – RR – 18/min, bilateral inspiratory crackles to mid zones, on 2l nasal cannula O₂ Sats 98% (dropped to 91% on room air)
    • C – well perfused, PR 68/min, BP 110/64, UO 10mls since midnight (7 L positive fluid balance)
    • D – alert
    • E – T 37 °C oedema – Nurse commented on swelling
Clinical Case

- **Invx**
  - Cr 275 ummol/L
  - K 4.8 mmol/L
  - Bicarbonate 19 mmol/L
  - CRP 126 (normal <5)

- **Plan**
  - STOP fluids (**Chasing the Oliguria**)
  - Septic screen
  - CXR
  - No place for Furosemide
  - If patient’s CXR demonstrates pulmonary oedema refer ICU for dialysis (continuous RRT)
Clinical Case

• **NEWS 3**
  – low risk????

• BUT

• No Urine Output

• AKI Stage 3

• Patient is at high risk

• What role is NEWS playing – counterbalance

• AKI 3 should be part of NEWS which mandated actions
Clinical Case

• Patient transferred to ICU
  – Cr 485 umol/L
  – K 6.2 mmo/L
  – Bicarbonate 14 mmol/L
  – CRP 274
  – Renal replacement therapy

• Consequences?
  – ↑ mortality
  – ↑ length of stay
  – chronic kidney disease (cardiovascular risk)
Clinical Case

- Patient required 24 hrs of RRT on ICU
- Started to recover kidney function
- Transferred back to orthopaedic ward
- Could this episode have been avoided?
Fluid Balance – knowledge gap

• currently there is a significant knowledge gap with respect to
  – volume status assessment
  – prescription of fluids

• and in the case of oliguric AKI
  – when to stop fluids and prevent volume overload and pulmonary oedema
Prevention of AKI

This also includes preventing the progression of AKI
The primary aim of the NHS England ‘Think Kidneys’ AKI Programme:

•...to ensure avoidable harm related to AKI is prevented in all care settings...
Patient Safety Alert

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

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

Actions
When: NHS England and trusts...
Automated, electronic alerts for acute kidney injury: a single-blind, parallel-group, randomised controlled trial

F Perry Wilson, Michael Shashaty, Jeffrey Testani, Iram Aqeel, Yuliya Borovskiy, Susan S Ellenberg, Harold I Feldman, Hilda Fernandez, Yevgeniy Gitelman, Jennie Lin, Dan Negoianu, Chirag R Parikh, Peter P Reese, Richard Urbani, Barry Fuchs
Tackling acute kidney injury – a multi-centre quality improvement project

Will test scalability and effectiveness of a package of interventions:

1. AKI detection and alerting
2. Education programme (hospital wide)
3. STOP AKI Care bundle

Stepped wedge design

Outcome measures: implementation, process, patient outcomes, balancing measures

Partner organisations:
Leeds Teaching Hospitals Trust
Derby Hospitals (lead organisation)
Bradford NHS Foundation Trust
Frimley Park Hospital
Ashford and St Peters Hospital
UK Renal Registry
Prevention of AKI

RISK FACTORS
• Clinical history
• Electronic patient record

PRESCRIBING
• Avoid Nephrotoxins

STOP AKI PREVENTION PATHWAY
• High-risk patients

ELECTRONIC DETECTION SYSTEMS
• Serum creatinine
• NEWS

Acute Kidney Injury (AKI)
Preventing AKI

Raising Awareness of AKI and Improving Education in the UK

Multi-Professional Approach
ACUTE KIDNEY INJURY: A COMPETENCY FRAMEWORK

www.aomrc.org.uk

DEFINING THE ROLE OF THE CLINICIAN

NOVEMBER 2011
Acute kidney injury: Prevention, detection and management of acute kidney injury up to the point of renal replacement therapy

NICE guidelines [CG169]  Published date: August 2013

Acute kidney injury

This clinical guideline offers evidence-based advice on the prevention, detection and management of acute kidney injury up to the point of renal replacement therapy.

More on this guideline

NICE article

New NICE kidney guideline to save thousands of lives
Prevention of Contrast Induced Acute Kidney Injury (CI-AKI) In Adult Patients

on behalf of

The Renal Association,
British Cardiovascular Intervention Society and
The Royal College of Radiologists

Dr Andrew Lewington, Consultant Renal Physician
Preventing CI-AKI

• Is study necessary?
• Identify patient at risk
• Review medications
  – avoid NSAIDs
• Volume expand
  – 0.9% sodium chloride 1 ml/kg/hr 12 hrs pre and 12 hrs post
• Minimise volume of contrast
• No place for N-acetylcysteine
• Repeat U&E 48hrs post procedure
CONSENSUS GUIDELINES FOR THE SAFE PRESCRIPTION AND ADMINISTRATION OF ORAL BOWEL CLEANSING AGENTS

Produced by:

Dr Andrew Connor, SpR in Nephrology, Dorset County Hospital, Dorchester.
On behalf of The Renal Association.

On behalf of the British Society of Gastrointestinal and Abdominal Radiologists for the Royal College of Radiologists

Dr Steve Hughes, Consultant Gastroenterologist, Southmead Hospital, Bristol.
On behalf of the Endoscopy Committee of The British Society of Gastroenterology.

Mr Nick Carr, Consultant Colorectal Surgeon, Singleton Hospital, Swansea.
On behalf of The Association for Coloproctology of Great Britain and Ireland for The Royal College of Surgeons.
**ORAL BOWEL CLEANSING AGENT PRESCRIPTION CHECKLIST**

This checklist is to be completed by the clinician authorising the oral bowel cleansing agent and should then be filed in the patient's medical records.

### STEP 1: ABSOLUTE CONTRAINDICATIONS

<table>
<thead>
<tr>
<th>Condition</th>
<th>Y/N</th>
</tr>
</thead>
<tbody>
<tr>
<td>GI Obstruction, ileus or perforation</td>
<td></td>
</tr>
<tr>
<td>Severe IBD</td>
<td></td>
</tr>
<tr>
<td>Toxic megacolon</td>
<td></td>
</tr>
<tr>
<td>Reduced conscious level</td>
<td></td>
</tr>
<tr>
<td>Hypersensitivity to any ingredients</td>
<td></td>
</tr>
<tr>
<td>Dysphagia (unless via NGT)</td>
<td></td>
</tr>
<tr>
<td>Ileostomy</td>
<td></td>
</tr>
</tbody>
</table>

If yes to any question, do not continue.

### STEP 2: REVIEW ANY BLOOD RESULTS

(Should be checked in patients with comorbidities)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na</td>
<td></td>
</tr>
<tr>
<td>K</td>
<td></td>
</tr>
<tr>
<td>eGFR</td>
<td></td>
</tr>
</tbody>
</table>

**STEP 3: Review MEDICATIONS**

<table>
<thead>
<tr>
<th>Medication</th>
<th>Y/N</th>
<th>Safe to stop for hrs?</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACEi/ARB</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diuretics</td>
<td></td>
<td></td>
</tr>
<tr>
<td>NSAIDs</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### STEP 4: CONSIDER CO-MORBIDITIES & RISK FACTORS

<table>
<thead>
<tr>
<th>Comorbidities</th>
<th>Optimal</th>
<th>Acceptable</th>
<th>Avoid</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kidney Disease</td>
<td>PEG / Pico / CF / Citramag</td>
<td>Pico / CF</td>
<td>OSP</td>
</tr>
<tr>
<td>CKD 3</td>
<td>PEG (if fluid status allows)</td>
<td>Pico / CF</td>
<td>OSP</td>
</tr>
<tr>
<td>CKD 4</td>
<td>PEG (if fluid status allows)</td>
<td>Pico / CF</td>
<td>OSP</td>
</tr>
<tr>
<td>Haemodialysis</td>
<td>Discuss with nephrologist</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peritoneal dialysis</td>
<td>Discuss with nephrologist</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Renal Transplant</td>
<td>Discuss with nephrologist</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Electrolyte Imbalance</td>
<td>PEG</td>
<td>Pico / CF</td>
<td>OSP</td>
</tr>
<tr>
<td>Cardiac Failure</td>
<td>PEG</td>
<td>Pico / CF</td>
<td>OSP</td>
</tr>
<tr>
<td>Liver Cirrhosis</td>
<td>PEG</td>
<td>Pico / CF</td>
<td>OSP</td>
</tr>
<tr>
<td>Hypertension</td>
<td>PEG / Pico / CF / Citramag</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### STEP 5: TYPE OF BOWEL PREP ISSUED?

- Picolax / Citrafleet / Citramag / Klean Prep / Moviprep / Fleet Phospho-soda

### STEP 6: INSTRUCTIONS PROVIDED TO THE PATIENT

<table>
<thead>
<tr>
<th>Method</th>
<th>Y/N</th>
</tr>
</thead>
<tbody>
<tr>
<td>Verbally</td>
<td></td>
</tr>
<tr>
<td>Leaflet</td>
<td></td>
</tr>
</tbody>
</table>

### STEP 7: OTHER COMMENTS

### STEP 8: SIGNATURE

**KEY**

- ACEi Angiotensin converting enzyme inhibitors
- ARB Angiotensin II Receptor Blockers
- CKD chronic kidney disease
- OSP oral sodium phosphate preparations (Fleet Phospho-soda)
- PEG polyethylene glycol (Klean Prep, Moviprep, Picolax, Citrafleet, Citramag)
Treatment of Hyperkalaemia
Emergency Management of Hyperkalaemia in Adults

Hyperkalaemia (K⁺ ≥ 5.5 mmol/L)
Consider initiating treatment if Hyperkalaemia suspected and K⁺ unknown

Airway Breathing Circulation Disability Exposure (ABCDE) Approach
Seek expert help if airway, breathing or circulation compromised

Assess Patient

Perform 12-lead ECG

Hyperkalaemia

MILD
K⁺ 5.5 - 5.9 mmol/L
Consider cause and if treatment indicated

Moderate
K⁺ 6.0 - 6.4 mmol/L
Treatment guided by clinical scenario, ECG and rate of rise

Severe
K⁺ ≥ 6.5 mmol/L
Emergency treatment indicated

Monitor ECG in high dependency area
Sick patient, K⁺ ≥ 5.5 mmol/L; Acute ECG changes present

NO

Acute ECG changes present (pick if present)?
- Peaked T waves
- Absent or flattened P waves
- Broad QRS
- Sine wave
- Bradycardia
- VT

YES

Calcium Chloride OR Calcium Gluconate IV
Repeat ECG and consider further dose after 5 min if ECG changes persist

Insulin–Glucose IV Infusion
Give in severe hyperkalaemia
Consider in moderate hyperkalaemia (assess ECG and rate of rise)

Salbutamol 10-20 mg Nebulised
Give in severe hyperkalaemia
Consider in moderate hyperkalaemia (assess ECG and rate of rise)

Remove K⁺ from body

Consider Dialysis
Seek advice from Renal or ICU team; patient transfer may be required

Consider cause of hyperkalaemia, prevent further rise and recurrence
Stop all nephrotoxic medication including ace-inhibitors, angiotensin II receptor blockers, potassium-sparing diuretics, NSAIDS and assess diet

Shift K⁺ into cells

Protect the Heart

Next 30-60 min

Glucose (25 g) over 15 min
50 ml 50% Glucose OR
125 ml 20% Glucose, WITH Soluble Insulin – 10 units
Salbutamol
Give 10 mg if history of IHD
Avoid if tachyarrhythmia present

Blood Monitoring:
Baseline
Glucose
K⁺
15 min
Glucose
K⁺
30 min
Glucose
K⁺
60 min
Glucose
K⁺

After 1 hour

Blood Monitoring:
90 min
Glucose
K⁺
120 min
Glucose
K⁺
180 min
Glucose
K⁺
240 min
Glucose
K⁺
360 min
Glucose
K⁺
24 hours
K⁺

K⁺: potassium; Na⁺: sodium; Creat: creatinine; IV: intravenous; min: minutes; PR: per rectum; EWS: early warning score; IHD: Ischaemic Heart Disease; NSAIDS: non-steroidal anti-inflammatory drugs
Education
Welcome
The philosophy of the RRAPiD programme is to emphasise timely and rapid response to the acutely ill patient and equip medical students and junior doctors with the appropriate skills to manage such patients. A specialist team of consultant physicians and clinical educators have developed the RRAPiD app, eBook and teaching programme to provide mobile and interactive resources suitable for this purpose.

Award
RRAPiD was shortlisted for the Times Higher Education Awards 2014 in the Outstanding ICT Initiative of the Year category.

Resources
RRAPiD eBook

Get the RRAPiD eBook via the App Store for iOS.
View RRAPiD eBook V1.3 online

RRAPiD iPhone App

Get the RRAPiD app via the App Store for iOS and via Google Play for Android.
RRAPID eBook and iPhone app

App Store FREE

eBook at rrapid.leeds.ac.uk/ebook

AKI Risk Calculator

NEWS calculator - RCP
Acute care toolkit 12
Acute kidney injury and intravenous fluid therapy September 2015
<table>
<thead>
<tr>
<th>STOP AKI</th>
<th>Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sepsis</td>
<td>&gt; Sepsis Six care bundle:</td>
</tr>
<tr>
<td></td>
<td>~ blood cultures</td>
</tr>
<tr>
<td></td>
<td>~ urine output – hourly</td>
</tr>
<tr>
<td></td>
<td>(urea and electrolytes)</td>
</tr>
<tr>
<td></td>
<td>~ fluids – IV</td>
</tr>
<tr>
<td></td>
<td>~ antibiotics – IV</td>
</tr>
<tr>
<td></td>
<td>~ lactate and haemoglobin</td>
</tr>
<tr>
<td></td>
<td>~ oxygen – high flow</td>
</tr>
<tr>
<td></td>
<td>&gt; Identify and treat source of sepsis</td>
</tr>
<tr>
<td>Toxins</td>
<td>&gt; Stop/avoid potential nephrotoxins:</td>
</tr>
<tr>
<td></td>
<td>~ gentamicin</td>
</tr>
<tr>
<td></td>
<td>~ NSAIDs</td>
</tr>
<tr>
<td></td>
<td>~ iodinated contrast</td>
</tr>
<tr>
<td>Optimise BP</td>
<td>&gt; Volume status assessment:</td>
</tr>
<tr>
<td></td>
<td>~ IV fluids</td>
</tr>
<tr>
<td></td>
<td>&gt; Hold BP-lowering medication</td>
</tr>
<tr>
<td></td>
<td>&gt; Consider vasopressors</td>
</tr>
<tr>
<td>Prevent harm</td>
<td>&gt; Treat complications</td>
</tr>
<tr>
<td></td>
<td>&gt; Identify the cause and investigate</td>
</tr>
<tr>
<td></td>
<td>&gt; Review:</td>
</tr>
<tr>
<td></td>
<td>~ all medications</td>
</tr>
<tr>
<td></td>
<td>~ fluid management plan</td>
</tr>
</tbody>
</table>
Sepsis 6

- Blood cultures
- Urine Output, U&E, Urinalysis
- Fluids
- Antibiotics
- Lactate/Haemoglobin
- Oxygen
Patient Education
**ACUTE KIDNEY INJURY RISK CARD**

- You have been given this card as you are **at risk** of acute kidney injury (AKI)
- Show this card to the pharmacist when buying or collecting any medication
- If you are admitted to hospital show this card to the doctors and nurses

<table>
<thead>
<tr>
<th>Poor fluid intake</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vomiting/diarrhoea</td>
</tr>
<tr>
<td>Infection</td>
</tr>
<tr>
<td>Dark concentrated urine</td>
</tr>
<tr>
<td>Decreased urine output</td>
</tr>
</tbody>
</table>

If you notice any of the below refer to the other side of the card

*If the holder of this card becomes acutely ill or is admitted to hospital, check the kidney function as he/she is at risk of ACUTE KIDNEY INJURY*
STOP ACUTE KIDNEY INJURY

THINK FLUIDs

**FLuids**
- Are you drinking enough fluid?

**Urine**
- When did you last pass urine?

**Infection**
- Do you have an infection?
  - If yes then you may need to see your GP

**Drugs**
- Are you on blood pressure tablets?
  - If yes
    - Do you feel light headed?
    - Is your blood pressure low?
  - If yes consult your GP
The Future

• NHS England AKI Risk calculators
  – primary and secondary care

• Biomarkers
  – HTA funded project in Leeds evaluating the role of biomarkers in the ICU
Conclusions

• AKI is a medical emergency
  – sepsis/IV fluids
• AKI can be prevented in some cases
• NEWS does not identify patients with AKI and can falsely reassure
• AKI risk assessment is a dynamic process
• STOP AKI can be used for prevention and management
‘Kidneys are for Life’

‘Save a nephron’
Thank You