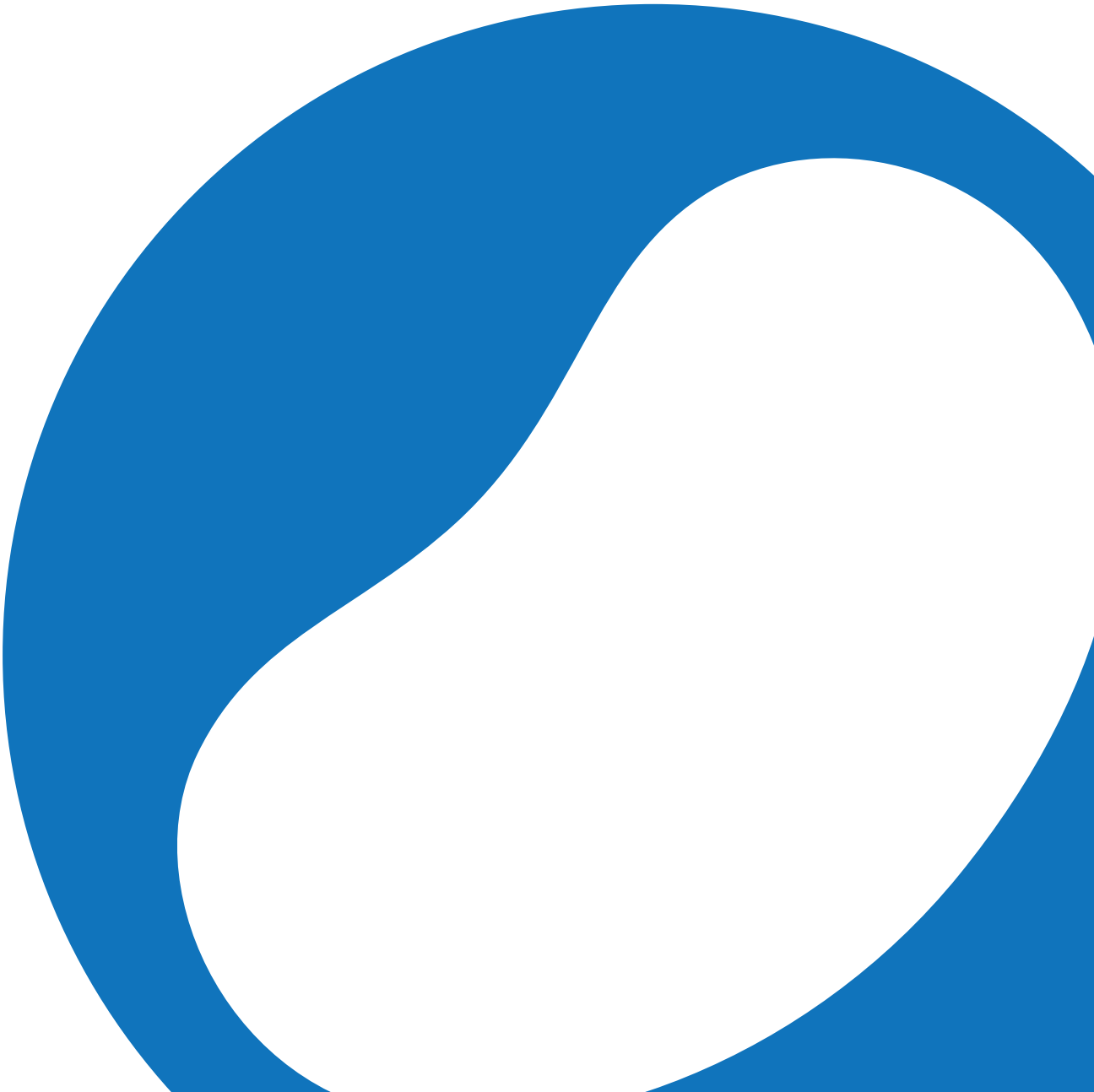
London Acute Kidney Injury Network

NHS
London
Strategic Clinical Networks

London AKI Network Manual

2.0 (2015)



Supporting the delivery of equitable, high quality AKI care through collaboration

We are part of london strategic clinical networks
We work with think kidneys
We were funded by Health Education North Central and East London

This information also available at londonaki.net and on our londonaki app for Android, iPad and iPhone

Hospital guidelines databases and e-alert notifications can be linked to www.londonaki.net/clinical

Full supporting educational resources at: www.londonaki.net/academy

Twitter: @londonaki

The London AKI Manual 2.0 (2015) contents have been updated in line with the following recent guidance

NICE Clinical Guideline 169 (2013)
NICE Clinical Guideline 174 (2014)
NICE Quality Standard 76 (2014)
Joint UK Renal Association, Royal College of Radiologists and British Cardiovascular Intervention Society guidance on prevention of contrast-induced acute kidney injury (CI-AKI) in adult patients (2014)
NHS England Stage 3 National Patient Safety Alert: Standardising the early identification of acute kidney injury (2014)
London Health standards on inter-hospital transfers 2014

The manual synthesises other available National guidance (referenced). Its contents were informed in consultation with network clinical leads across London in 2012. Updates have been ratified by the London AKI Network Board

Updates will be reviewed every three years on the basis of new national guidance or emergent evidence

Overview

- The manual collates available evidence, national guidelines and clinical standards into clear AKI patient pathways and accessible, practical advice. It is designed for those managing AKI in general ward areas. It also aims to clarify the interaction between general wards, local critical care and regional kidney unit services. The availability of a written guideline with this content is an NCEPOD standard for each NHS Trust.
- Use of the guidelines and manual is optional. Trusts may operationalize the manual contents as a Trust guideline or adapt its contents to local needs. Trusts are encouraged to link guideline databases to our clinical guideline (http://www.londonaki.net/clinical) and users may access our general educational resources on www.londonaki.net/academy
- We will guarantee that this guideline will be quality assured, updated on a three yearly basis and that the feedback of network members will inform its development.
- Definitions of AKI have not changed. AKI is present if there has been a 1.5 rise in serum creatinine from a presumed 7 day baseline or a >26micmol/l rise within 48 hours. The National AKI detection algorithm will produce a 'test message' incorporating an annualised median baseline, however clinicians must use discretion in interpreting such test messages.
- The AKI care bundle (management and investigation) should be instituted for all patients classified as AKI i.e. 1.5 x rise from the most recent baseline Cr (in the last year) or 6 hours of oliguria (0.5mls/kg/hr). While the bundle may be instituted earlier (e.g. for creatinine rises of >26micmol/ml) we recommend pathway activation as a basic standard of care for patients who have even mild AKI at this threshold.
- Patients who progress to, or have, AKI 3 which represents >80% loss of kidney function, should be discussed with the local nephrology (2013 NICE guidance) with the exception of multi-organ failure patients managed in critical care. We also recommend that such severe, ward-based AKI patients should be discussed with local critical care teams.
- Nephrology advice should be sought on any AKI patient unless the cause has been identified and that cause can be effectively treated by the base team.
- Patients with even mild AKI should be referred to nephrology services if primary renal disease is suspected (e.g. glomerulonephritis, tubulointerstitial nephritis, haemolytic uraemic syndrome). Such patients need specialist nephrology diagnosis (possibly including renal biopsy) and management.
- If transfer to critical care is required this should be as soon as possible. Transfer target to kidney unit is 24 hours, but there are currently heavy demands on acute renal bed useage at some sites.
- Patients with evolving multi-organ failure should be managed locally in critical care. They will generally not meet transfer safety criteria. Guidelines for this and who should be referred from ITU to renal are in the manual.
- The basic panel of investigations is USS, dipstick and routine haematology and biochemistry. More specialist tests (anti-GBM etc) may be done but results delivery should not delay the referral process.
- USS should be performed<24 hours for all non-recovering AKI where the cause is not obvious. The target is <6 hours where urinary obstruction with infection is suspected. (NICE 2013).
- In general single organ support should be provided within the regional renal unit. Some patients need stabilisation prior to transfer as outlined in the guideline. In some patients having on-going specialist care (e.g. complex surgery or cancer care) it may be preferable to manage the patient in the local ITU to maintain continuity with the base speciality teams.
- Temporary lowering of K with insulin and dextrose does not facilitate safe transfer (as there may be rebound in transit) and hyperkalaemic patients should have onsite CVVH or bicarbonate prior to transfer such that the K lowering is likely to be sustained.
- We would recommend early discussion with your nephrology or critical care teams when there is any uncertainty regarding the most appropriate clinical plan.
- These are guidelines rather than binding protocols. Guidelines inform and harmonise practice but are not a substitute for the proper clinical assessment of individual cases. We will guarantee that our materials represent consensus, National guidelines, available evidence and are up to date. We cannot assume clinical responsibility for the consequences of deployment of these guidelines, appropriately or otherwise.

Risk, Prevention and Recognition

Some AKI Is Predictable, Preventable and/or Recognised Late

Risk Assess for AKI
The risk of AKI is contributed to by the acute insult and background morbidity

Background
Elderly (>65)
CKD
Cardiac failure
Liver disease
Diabetes
Vascular disease
Background nephrotoxic medications

Acute 'STOP'
Sepsis and hypoperfusion
Toxicity
Obstruction
Parenchymal kidney disease

Prevent AKI - The 4 'M's
Monitor Patient
(observations and EWS, regular blood tests, pathology alerts, fluid charts, urine volumes)
Maintain Circulation
(hydration, resuscitation, oxygenation)
Minimise Kidney Insults
(e.g. Nephrotoxic medications (NSAID, aminoglycosides, ACE/ARB, diuretics), surgery or high risk interventions, iodinated contrast and prophylaxis, hospital acquired infection)
Manage The Acute Illness
(e.g. sepsis, heart failure, liver failure)

Recognise AKI
1.5 rise from recent baseline creatinine, >26 rise in 48 hours,
prompt from National algorithm or 6 hours of oliguria

AKI Develops

INSTITUTE CARE BUNDLE
Prevent AKI progression by rapid diagnosis, supportive care, specific therapy and appropriate referral

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AKI Care Bundle

Institute in all patients with a 1.5 x rise in creatinine or oliguria (<0.5mls/kg/hr) for 6 hours
(for 26.4 micmol/l rises activating National detection algorithm assess and consider institution or recheck)

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, **T**oxicity, **O**bstruction, **P**rimary 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

Follow up

Ensure patient/carers have adequate support and information
Monitor recovery to completion and ensure adequate follow up arrangements in place

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AKI Care Bundle Checklist

Patient Name: .....

No: ..... **DOB:** .....

URGENT ASSESSMENT	YES	NO	N/A
ABC and full set of observations	☐	☐	☐
Oxygen therapy?	☐	☐	☐
National early warning system triggering	☐	☐	☐
Critical care outreach called (if triggering)	☐	☐	☐

DIAGNOSE THE CAUSE(S)	YES	NO	N/A
Sepsis and hypoperfusion	☐	☐	☐
Toxicity	☐	☐	☐
Obstruction	☐	☐	☐
Primary renal disease	☐	☐	☐

TREAT THE CAUSE(S)	YES	NO	N/A
Bolus fluid to restore hypovolaemia	☐	☐	☐
Sepsis screening and antibiotics	☐	☐	☐
Severe sepsis antibiotics <1 hour and 'sepsis six'	☐	☐	☐
Relative hypotension stop antihypertensives	☐	☐	☐
Stop nephrotoxins (including ACE/ARB/NSAID)	☐	☐	☐
If obstruction confirmed referred urology	☐	☐	☐
Obstruction relieved	☐	☐	☐
If primary renal disease suspected referred nephrology	☐	☐	☐
If indicated therapy for renal disease given	☐	☐	☐

GENERAL SUPPORTIVE CARE AND ESCALATION	YES	NO	N/A
Maintenance fluid prescription and monitoring plan	☐	☐	☐
Physiological monitoring plan	☐	☐	☐
Maintenance drugs and dosages reviewed	☐	☐	☐
Monitoring blood tests arranged	☐	☐	☐

AKI REFERRAL AND ESCALATION	YES	NO	N/A
Referral pathway reviewed	☐	☐	☐
Referred nephrology (AKI 3, no recovery, complications cause unclear or primary renal disease)	☐	☐	☐
Referred local critical care (AKI 3, no recovery, complications)	☐	☐	☐

FOLLOW UP	YES	NO	N/A
Patient/carer adequate support and information	☐	☐	☐
Follow up arrangements in place and communicated to relevant clinicians	☐	☐	☐

Signed: .....

Date: .....

Position: .....

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'STOP' AKI and Checklist

The London AKI Network has Developed the 'STOP' Acronym to Improve Awareness of AKI Causes

STOPaki

Sepsis & hypoperfusion Toxicity Obstruction Primary renal disease

Patient Name: .....

No: ..... **DOB:** .....

SEPSIS & HYPOPERFUSION	YES	NO	N/A
Severe Sepsis	○	○	○
Haemorrhage	○	○	○
Dehydration	○	○	○
Cardiac Failure	○	○	○
Liver Failure	○	○	○
Renovascular Insult (E.G. Aortic Surgery)	○	○	○

TOXICITY	YES	NO	N/A
Nephrotoxic Drugs	○	○	○
Iodinated Radiological Contrast	○	○	○

OBSTRUCTION	YES	NO	N/A
Bladder Outflow	○	○	○
Stones	○	○	○
Tumour	○	○	○
Surgical Ligation Of Ureters	○	○	○
Extrinsic Compression (E.G. Lymph Nodes)	○	○	○
Retroperitoneal Fibrosis	○	○	○

PRIMARY RENAL DISEASE	YES	NO	N/A
Glomerulonephritis	○	○	○
Tubulointersitial Nephritis	○	○	○
Rhabdomyolysis	○	○	○
Haemolytic Uraemic Syndrome	○	○	○
Myeloma Kidney	○	○	○
Malignant Hypertension	○	○	○

Signed: .....

Date: .....

Position: .....

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AKI Complications

Hyperkalaemia, Acidosis, Pulmonary Oedema, Reduced Conscious Level

Begin Medical Therapy and Get Help

Local Critical Care Team
and
Local Nephrology Team (if onsite)

Hyperkalaemia

Medical therapy of hyperkalaemia is a transient measure pending imminent recovery in renal function or transfer to kidney unit or critical care for renal replacement therapy.
If ECG changes give calcium gluconate 10mls 10%.
If bicarbonate <22mmol/L and no fluid overload give 500mls 1.26% sodium bicarbonate over 1 hour.
K>6.5mmol/L or ECG changes give insulin 10 international units in 50mls of 50% dextrose over 15 minutes & salbutamol
10mg nebulised (caution with salbutamol in tachycardia or ischaemic heart disease).
Insulin/dextrose and salbutamol reduce ECF potassium for <4 hours only.

Acidosis

Medical therapy of acidosis with bicarbonate should be reserved fo emergency management of hyperkalaemia (as above) pending specialist help.
pH<7.15 requires immediate critical care referral.

Pulmonary Oedema

Sit the patient up and give oxygen (60-100% unless contraindicated)
If haemodynamically stable give furosemide 80mg IV. Consider repeat bolus and infusion at 10mg/hour
If haemodynamically stable commence GTN 1-10mg/hour titrating dose.

Reduced Conscious Level

Manage uraemic coma as per all reduced consciousness (airway management) pending critical care transfer and emergency renal replacement therapy.

These are Holding Measures Prior to Specialist Help from Critical Care or Nephrology Services

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Referral from Ward

All AKI
with
Blood or protein on dipstick
Possible autoimmune disease/ glomerulonephritis, myeloma
Possible HUS/TTP, hypertension
Poisoning.
Renal transplant and CKD stage 4/5

Local Renal Team
If transfer decided see AKI transfer policy

All AKI
with
Obstruction on USS
(NB partially obstructed patients may have normal or high urine volumes).

Local Urology Team
If nephrostomy or stenting required proceed immediately

Progression to AKI 3 Or AKI 3 at Recognition or AKI Complications and Imminent Recovery Unlikely

Local Renal Team
or
Local Critical Care Team
(essential if the patient is developing multi-organ failure)

If the Patient Is Too Ill To Transfer (see AKI Transfer Policy)
Contact Local Critical Care Team

Institute AKI Care Bundle While Transfer Pending

Dataset Needed for Kidney Unit Referrals
U and E, Calcium, Phosphate, ABG/lactate, FBC, coagulation, LFTs.
Heart rate, respiratory rate, blood pressure, Oxygen saturations.
AVPU or GCS score.
Urine output.
AKI grade and pre-morbid Cr level.
Urine dipstick.
USS if obtained.
Co-morbid history.
MRSA status (if known).

Referral from Ward to Kidney Unit Checklist

The following data are required for referral to your local renal service
Please use this checklist to ensure you have all this essential information
This checklist is also available on the London AKI iPhone App

Patient Name: .....

No: ..... **DOB:** .....

	YES	NO	N/A
Urea and Electrolytes	☐	☐	☐
Calcium	☐	☐	☐
Phosphate	☐	☐	☐
Arterial Blood Gases and Lactate	☐	☐	☐
Urine Dipstick	☐	☐	☐
USS Result (if performed)	☐	☐	☐
Baseline Renal Function (if known)	☐	☐	☐
Past Medical History	☐	☐	☐
Blood Pressure	☐	☐	☐
Heart Rate	☐	☐	☐
Oxygen Saturations	☐	☐	☐
Respiratory Rate	☐	☐	☐
Avpu or GCS Assessment of Conscious Level	☐	☐	☐
Current Urine Volume	☐	☐	☐
Mrsa Status	☐	☐	☐
Whether Diarrhoea Last 48 Hours	☐	☐	☐

Signed: .....

Date: .....

Position: .....

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Transfer From Ward to Kidney Unit
(interhospital transfer)

The following is a guideline for whether patients are safe to transfer from a ward to a kidney unit in another hospital.
All AKI patients for transfer should be assessed by a senior (ST4+) doctor.

Hyperkalaemia
No ECG changes.
K < 6.0mmol/L.
If K lowered to <6.0 after presentation this must be potentially sustained (e.g bicarbonate therapy or dialysis/CVVH) not transient therapy (insulin and dextrose).

Renal Acidosis
pH >7.2.
Venous bicarbonate >12mmol/L.
Lactate < 4mmol/L.

Respiratory
Respiratory rate >11 and < 26/min.
Oxygen saturations >94% on not more than 35% oxygen.
If patient required acute CPAP must have been independent of this treatment for 24 hrs.

Circulatory
Heart rate > 50/min and < 120/min.
Blood pressure > 100mmHg systolic.
MAP > 65MMHg.
Lactate < 4mmol/L.
(lower BP values may be accepted if it has been firmly established these are pre-morbid).

Neurological
Alert on AVPU score or GCS >12.

If Criteria not Met Emergency Referral to Local Critical Care
Once stabilised follow ITU to acute kidney unit transfer policy.

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Transfer from Ward to Kidney Unit Checklist

The following is to enable renal teams to screen referrals for transfer safety
All AKI patients for transfer should be assessed by a senior (ST4+) doctor
This checklist is also available on the London AKI iPhone App

Patient Name: .....

No: ..... **DOB:** .....

	YES	NO	N/A
Potassium <6.0mmol/l			
pH>7.2			
Venous Bicarbonate >12mol/l			
Calcium (ionised > 1mmol/l, total > 2mmol/l)			
Lactate (< 4 mmol/l)			
Blood Pressure (>100mmHg)			
MAP (>65mmhg)			
Heart Rate (>50/min and <120/min)			
Oxygen Saturations (>94% on not more than 35% O2)			
Respiratory Rate (>11/min and <26/min)			
AVPU Alert or GCS > 12			
All AKI patients for transfer should be assessed by a senior (ST4+) doctor			
MRSA Status			
Whether Diarrhoea in Last 48 Hours			

Signed: .....

Date: .....

Position: .....

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Referral from Critical Care to Nephrology

For step down care see:
AKI transfers policy critical care to kidney unit

Requests for nephrology advice (not-transfer) on critical care patients should be made to liaison nephrologist for the hospital or, if unavailable, to local on-call renal team.

Referral for nephrology opinion is at the discretion of the consultant intensivist and generally not necessary in patients with AKI in the context of multi-organ failure.

Referral is recommended if

Possibility of AKI as an initiating event (with subsequent systemic decompensation) - i.e AKI 3 early in illness.
Single organ failure.
AKI with possible vasculitis, lupus or autoimmune disease.
AKI in myeloma or malignancy or tumour lysis.
AKI with unexplained pulmonary infiltrates or pulmonary haemorrhage.
HUS/TTP.
AKI in pregnancy.
AKI with urological abnormalities.
AKI with malignant hypertension.
AKI with poisoning.

Transfer From Critical Care to Kidney Unit
(interhospital transfer)

Phone Local Renal Team

If the Patient is Accepted for Transfer, a Handover to Critical Care in Receiving Hospital Should be Done and Critical Care Outreach Informed

Further discussion with receiving hospital intensivist not required if condition stable or improving

Below is a Guideline for What Would be Considered a Safe ITU to Kidney Unit Transfer. These Transfers Should be Discussed at a Senior Level.

Metabolic
K < 6.0, ionised Ca > 1mmol/L.
pH normal.
Bicarbonate > 16mmol/L.
Lactate normal.

Respiratory
Respiratory rate >11/min and < 26/min.
Saturations > 94% on not more than 35% oxygen.
If patient required acute CPAP must have been independent of this treatment for 24 hrs.
If ventilated <1 week should have been independent of respiratory support for 48hrs.
If longer term invasive ventilation should have been independent of all respiratory support for 1 day for each week ventilated and for a period of not less than 48 hours.

Circulatory
Heart rate > 50/min and < 120/min.
BP > 100mmHg systolic.
MAP > 65MMHg.
If given inotropes given must have been inotrope independent > 24 hours.

Neurological
Alert AVPU (unless stable, chronic neurological impairment).

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Referral from Primary Care

AKI 3 at recognition (creatinine 3 x baseline)

Local Renal Team

Direct Admission to Kidney Unit for Assessment

AKI 2 at recognition (creatinine between 2 and 3 x baseline)

Local Acute Medical Team

Follow AKI Care Bundle and Referral Guideline

AKI 1 at recognition (creatinine between 1.5 and 2 x normal)

Follow Primary Care AKI Bundle

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Contrast Induced Nephropathy (CIN) Prophylaxis

Assess Risk

High volume (100mls) or intra-arterial iodinated contrast procedure
and
CKD with eGFR<60 (particularly diabetic nephropathy)
***or* AKI**
Other risk factors >75, dehydration, heart failure, severe sepsis, cirrhosis, nephrotoxins (NSAIDS, aminoglycosides). High volume or arterial contrast Risk factors are multiplicative.

Is Contrast Procedure Necessary?

Yes

Resuscitate to euvolaemia

Give Prophylaxis if High Risk
Volume expansion (unless hypervolaemic) with normal saline or or 1.26% bicarbonate
Sample regimens
IV Na bicarbonate 1.26% 3mls/Kg/hr for 1 hour pre-procedure and 6 hours post-procedure
or
IV 0.9% normal saline 1ml/kg/hr 12 hours pre and 12 hours post procedure

Minimise contrast, use low or iso-osmolar contrast

Monitor Function To 72 Hours in High Risk Cases
If oliguria or rising creatinine early referral to local renal team.
NB there is no-proven role for N-Acetyl cysteine or post-contrast dialysis/CVVH.
Cessation of metformin should be considered if serum Cr above reference range or eGFR<60.
Cessation of ACE inhibitors should be considered if acutely ill.

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Perioperative AKI

Preoperative AKI Risk Assessment
(anaesthetic and surgical teams) in pre-assessment clinic or ward

ASA score, consider pre-operative CPEX testing.
Pre-morbid factors: 65 years old, CKD, diabetes, vascular disease, cardiac failure, liver failure.
In emergency surgery consider current patient stability/illness severity.
Type of surgery: If 'major' operation or known high risk (e.g. cardiac bypass, intraperitoneal surgery, likely heavy blood loss or involving pelvis or renal tract).
Risk of perioperative nephrotoxic medications.

Consider pre-optimisation in ward or critical care area and scheduled post-operative admission to critical care.
There is no role for the routine use of dopamine or frusemide in perioperative AKI prevention.
DIscontinue or avoid nephrotoxic drugs if possible.
If risk of long-term renal insufficiency (e.g. nephrectomy in CKD discuss with nephrology team).
Optimise circulation and oxygenation during surgery.

Postoperative AKI Risk Assessment

As per pre-op assessment. Assess surgery undertaken, blood loss, perioperative haemodynamic stability, perioperative oxygenation and perioperative oliguria.

Monitor
Observations (blood pressure, heart rate, urine volumes, regular blood tests)

Postoperative resuscitation as appropriate

If postoperative AKI develops
Institute AKI Care Bundle and Referral Pathway

Consider and Treat Specific Surgical Causes
Blood loss, hypovolaemia, surgical sepsis, hypotension due to epidural or opiate anaesthesia, postoperative urinary retention or obstruction of the renal tract as a surgical complication.

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Fluids

Adult Maintenance Fluids

Baseline Requirements
50-100mmol sodium, 40-80mol potassium
and 1.5-2.5L water per 24 hours
Oral, enteral or parenteral route

Adjust estimated requirements according to changes in sensible or insensible losses

Sensible Losses
(measurable)
Surgical drains
Vomiting
Diarrhoea
Urine
(variable amounts of electrolytes)

Insensible Losses
Respiration
Perspiration
Metabolism
Increase in pyrexia or tachypnoea
(Mainly water)

Regular assessment of volume and hydration status
Daily weights
Fluid charts
Measured electrolytes

Available parenteral solutions (if required)
Hartmans solution/Ringer's lactate
Normal Saline
5% dextrose
0.4%/0.18% dextrose/saline
Potassium usually added additionally

Adult Resuscitation or Replacement Fluids

Give According to Clinical Scenario

General Volume Replacement or Expansion
Give balanced crystalloid solutions (Hartman's solution/Ringer's lactate)
These contain small amounts of potassium. Avoid in hyperkalaemia. If AKI only use these if close (HDU) monitoring of potassium
or
Colloids
Avoid high molecular weight (>200kDa starches in severe sepsis due to risk of AKI
Assess vital signs, postural blood pressure, capillary refill, JVP and consider invasive or non-invasive measurement using flow-based technology

Haemorrhage
Give blood and blood products
Balanced crystalloid or colloid may be given while blood awaited
Clinical assessment as above

Severe Free Water Losses (hypernatraemia)
5% dextrose
or 4%/0.18% dextrose/saline

Hypochloraemia
(vomiting, NG drainage)
Give normal saline
(Potassium repletion usually also required)

Obstetric AKI Pathway

Institute in all cases with creatinine >90µmmol/L or serial creatinine rise of 26µmmol/L or 20ml/hr urine for 12 hours (if PET excluded)

THIS IS POTENTIALLY A MEDICAL EMERGENCY

Full set of physiological observations BP/HR/RR/SATS/TEMP
Assess for signs of shock/hypoperfusion- low BP/high HR/confusion/pale & cold skin
Review history and past results If MEOWS triggering- high flow oxygen, Review senior/HDU/ITU

Fluid therapy in AKI

If hypovolaemic give crystalloid 250ml. Followed by 125 ml/hr* Re-assess
Catheterise if obstruction and measure hourly urine output

Monitoring in AKI

Venous blood gas & lactate, U&E twice a day while creatinine rising
Fluid chart, regular fluid assessment and observations

Investigations in AKI

If proteinuria URGENT PCR
Ultrasound (obstruction),
Liver Profile, If low platelets blood film (fragmented RBC/PLT), LDH, Bilirubin, Reticulocytes

Supportive AKI care

Sepsis- ANTIBIOTICS within an hour. Review drug chart/thromboprophylaxis

Causes Think STOP AKI
Prerenal Sepsis/hypovolaemia (PPH)
Renal Toxicity NSAIDS, PET, HELLP, HUS,TTP
Postrenal Obstruction or ureteric damage during delivery

* Caution with PET

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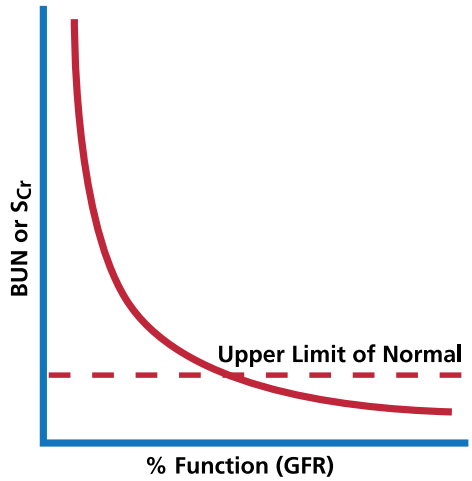
AKI Teaching Materials

"STOP" - causes of AKI

Sepsis and hypoperfusion (hypovolaemia, heart failure, hepatorenal)
Toxicity (drugs, contrast)
Obstruction
Parenchymal kidney disease (myeloma, rhabdomyolysis, RPGN, HUS, TIN)

Rising creatinine = rising mortality



KDIGO Staging System for Acute Kidney Injury

Stage	Serum creatinine	Urine output
1	rise ≥ 26 µmol/L within 48hrs or rise ≥1.5- to 1.9 X baseline SCr	<0.5 mL/kg/hr for > 6 consecutive hrs
2	rise ≥ 2 to 2.9 X baseline SCr	<0.5 mL/kg/hr for > 12 hrs
3	rise ≥3 X baseline SCR or rise 354 µmol/L or commenced on renal replacement therapy (RRT) irrespective of stage	<0.3 mL/kg/hr for > 24 hrs or anuria for 12 hrs

website: www.londonaki.net
email: info@londonaki.net

Kidney Unit Contact

North Central London Network Hospitals

UCL Centre for Nephrology Royal Free, Royal Free Hospital
AKI registrar mobile 07908422116
or 0207 794 0500 bleep 2608 via switchboard

North East London Network Hospitals

Bart's Health Renal Unit
Renal registrar via switchboard 0203 594 0000

North West London Network Hospitals

Imperial Renal and Transplant unit
Hammersmith Hospital
Telephone 0203 313 1000, renal registrar on-call, bleep 9977 or via switchboard

South East London Network Hospitals

Guy's and St Thomas' Renal Unit, Guy's Hospital
AKI registrar mobile 07789505184 or 0207 188 7188 renal registrar on-call via switchboard
King's Renal Unit, King's College Hospital
Telephone 0203 299 9000, renal registrar on call, bleep 622 or wifi extension 37716
Lead nurse for AKI contact 0203 299 5411

South West London Network Hospitals

South West Thames Renal and Transplantation Unit, St Hellier
Please use refar a patient.org for referrals to St Hellier renal unit. If urgent please call 0208 296 2000 renal registrar bleep 665
St George's Renal Unit, St George's Hospital
Telephone 0208 672 1255, renal registrar on-call, bleep 6415 or via switchboard

Recognition

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Recognition

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