

ACUTE KIDNEY INJURY (AKI) GUIDELINES

Reference no.: CG-T/2013/123

These guidelines cover prevention, detection and management of acute kidney injury (AKI). They aim to improve assessment and detection by non-specialist clinicians and specify when they should refer patients to specialist services. This will reduce the chance of death or complications for people at risk of acute kidney injury. **They are not a substitute for clinical judgement.**

- Acute kidney injury is a clinical syndrome characterised by a rapid reduction in kidney excretory function, and is associated with poor clinical outcomes. Acute kidney injury (AKI) is not a single condition, but has a variety of different causes.
- Acute kidney injury is seen in 13–18% of all people admitted to hospital, with older adults being particularly affected. Up to 30% of cases of acute kidney injury may be preventable.
- The international guideline group Kidney Disease: Improving Global Outcomes (KDIGO) has defined AKI according to **rises in serum creatinine and/or reductions in urine output**.
- Any patient who meets the criteria for AKI should be **reviewed to ascertain the cause of AKI and the severity of the injury should be staged**. Patients with more severe AKI are at greater risk of adverse outcomes, including progression to chronic kidney disease (CKD).

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PREDICTION & RECOGNISED RISK FACTORS OF AKI

All patients should be assessed on admission (within 24 hours) for the risk of developing AKI. At the Royal Derby Hospital this assessment is found on the Lorenzo system (Figure 1) and which **must be completed for all emergency admissions**. A score of ≥ 5 indicates high risk. At Queens Hospital Burton there is only the AKI care bundle on Meditech and not an AKI Risk Score tool.

All patients at high risk of AKI should have additional measures to try and prevent and detect AKI. These measures are :

1. Perform a fluid assessment and correct hypovolaemia
2. Review medications and withhold those that may increase risk of AKI (see below for list of medications)
3. Order daily U/Es so that if AKI does occur it will be more reliably detected

The screenshot shows the AKI Risk Score tool interface. It includes the following sections and questions:

- AKI Risk Score**
 - Patient not applicable
 - Age >75: Yes No
- Clinical**
 - Hypotension: Systolic BP >100mmHg or more than 40mmHg less than known baseline: Yes No
 - Sepsis (*SIRS=2 or more of the following: Temp <36 or >38, pulse >=90, WBC <4 or >12, RR>20 or PCO2 <4): Yes No
 - Hypovolaemia (include h/o diarrhoea, vomiting, increased fluid losses or reduced oral intake): Yes No
- Nephrotoxins**
 - ACE Inhibitors, ARBs, NSAIDs, diuretics, contrast: Yes No
- Comorbidities**
 - Click here to view currently recorded problems
 - Evidence of CKD eGFR <60ml/min/1.73m2: Yes No
 - Past history of CCF or current presentation consistent with acute cardiac failure: Yes No
 - Diabetes mellitus: Yes No
 - Atherosclerotic vascular disease: Yes No
 - Cirrhosis of liver: Yes No
- Total AKI Risk Score**: [Input field]
- Please click on the links to view guidance**
 - [AKI Clinical Guidelines Koha](#)
 - [AKI information and links Net-](#)

Figure 1 Screenshot of AKI Risk score tool on Lorenzo

CRITERIA FOR RECOGNISING AND STAGING AKI

- The AKI staging system is based on change in serum creatinine and urine output. If these lead to different AKI stages, use the highest.

Stage	Serum creatinine	Urine output
1	Increase in serum creatinine of $>26\mu\text{mol/L}$ from baseline within a 48hr period or Increase of 1.5 to 1.9 times baseline	$< 0.5 \text{ mL/kg/hour}$ for > 6 hours
2	Increase in serum creatinine of 2 to 2.9 times baseline	$< 0.5 \text{ mL/kg/hour}$ for > 12 hours
3	Increase in serum creatinine to 3 times baseline or Increase in serum creatinine to $>354\mu\text{mol/L}$ or Initiation of renal replacement therapy	$< 0.3 \text{ mL/kg/hour}$ for > 24 hours or no urine output > 12 hours

- Lorenzo and Meditech will automatically issue reports (**
- Figure 2 and Figure 3) on all patients who sustain AKI by creatinine criteria. These reports only take account of changes in creatinine and it is up to you to consider changes in urine output.
- Baseline creatinine is calculated as per the NHS England algorithm: either the lowest value over the last 7 days, or a median of values from the prior 7-364 days will be used depending on availability of previous results. (see <https://www.england.nhs.uk/wp-content/uploads/2014/06/psa-aki-alg.pdf> and <https://www.thinkkidneys.nhs.uk/wp-content/uploads/2014/12/AKI-Warning-Algorithm-Best-Practice-Guidance-final-publication-0112141.pdf> for more information). When a previous value is not available, the clinician should make a decision whether patient had AKI or CKD depending on further information if available from other laboratory results or further subsequent investigation. **In all cases, AKI remains a clinical diagnosis** – the serum creatinine and AKI result need to be interpreted within the clinical scenario. Sometimes, it is difficult to tell immediately if a patient has AKI - repeating the creatinine to look for subsequent acute change and taking account of the clinical picture may help.
- The laboratory team at RDH will phone the clinical areas, once, with any new AKI 2 or 3 alerts.
- PLEASE DOCUMENT “AKI” IN CLINICAL NOTES AND STATE THE CAUSE FOR THE AKI ALONG WITH ANY COMPLICATIONS



Figure 2 Screenshot of Blood results showing AKI stage on Lorenzo

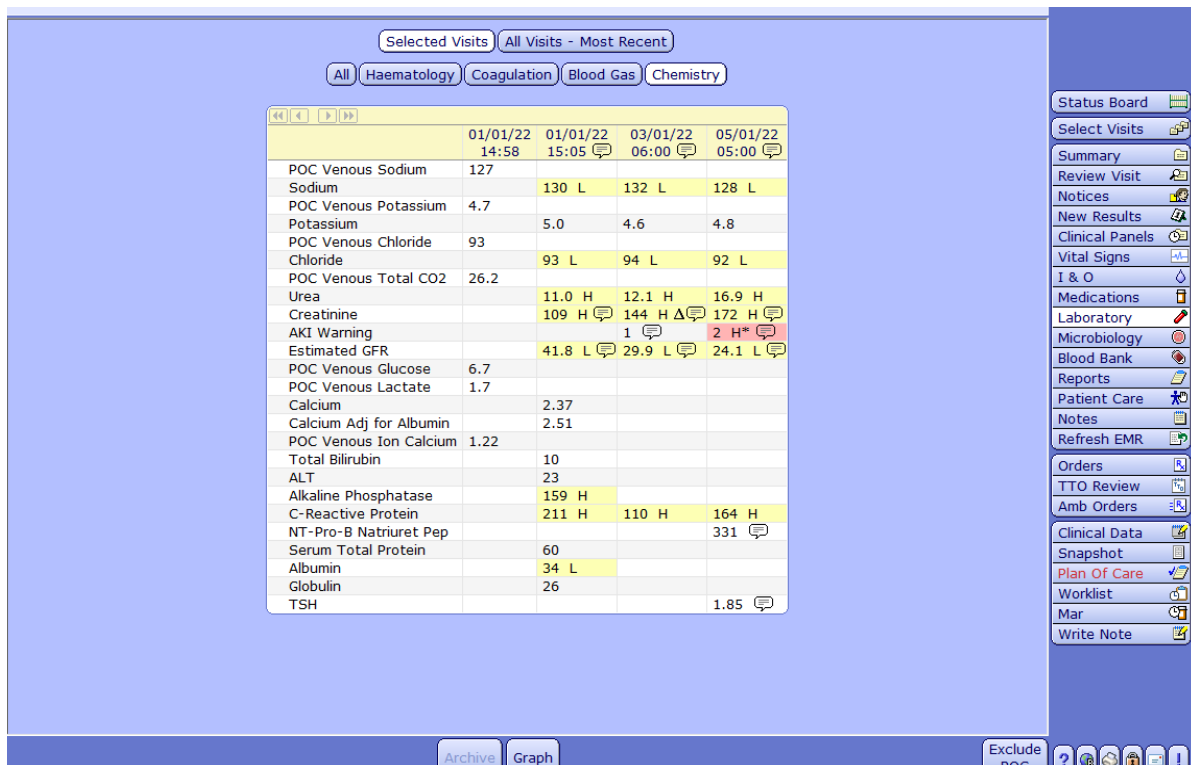
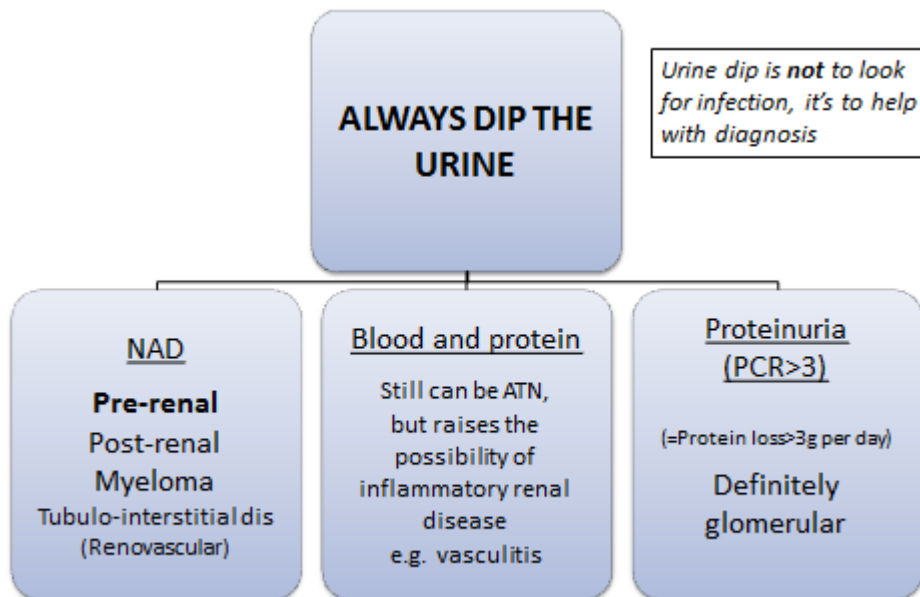


Figure 3 Screenshot of Blood results showing AKI warning/stage in Meditech

INITIAL INVESTIGATIONS IN AKI

- **History & Clinical Examination**
 - Patients with AKI often present with other acute illness. AKI should be managed alongside other conditions, in particular sepsis, but it indicates that the patient is at higher risk of deterioration.
 - **The most important factors relating to AKI that should be determined are:**
 - Clinical diagnosis of cause of AKI
 - Assessment of volume status.
- **Bloods** UE, Bicarbonate, FBC, LFT, Ca^{2+} / PO_4^{-3} Blood cultures (if sepsis suspected)
- **Urinalysis**

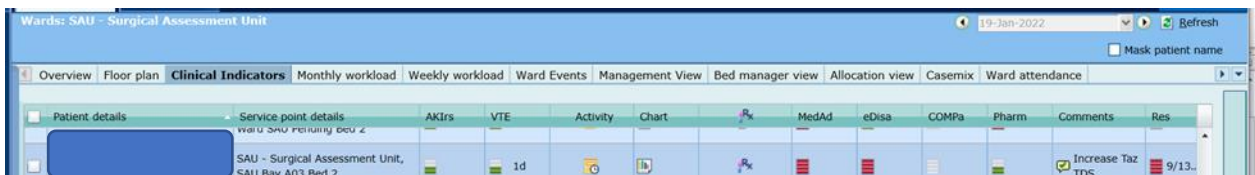


- **Ultrasound scan** of renal tract and bladder in patients suspected with;
 - Obstruction, scan within 24 h
 - Pyonephrosis, scan within < 6 h
 - Non-resolving/persistent AKI
- Other investigations should be guided by clinical findings. Examples include:
 - If platelets are low, request blood film, LDH (in case of haemolytic uraemic syndrome/ thrombotic thrombocytopenic purpura)
 - Creatine kinase (if rhabdomyolysis is suspected)
 - Immunology screen (ANA, ANCA, anti-GBM) if vasculitis is suspected
 - Myeloma screen if calcium is high

AKI CARE BUNDLE

If your patient has an AKI, the care bundle (Figure 4 on Lorenzo) (Figure 5 in Meditech) should be followed and completed as soon as possible after recognition of AKI. The care bundle is to help ensure that some of the most important aspects of AKI management are performed; it is not a complete care plan and other aspects of AKI care may still be required. **There is now evidence that completing the actions in the care bundle improves outcomes for patients.**

The AKI care bundle form can be found in the Forms screen in Lorenzo or via the ward clinical indicator screen. The icon in the activity column highlights with a clock if a care bundle requires completing and can be used to initiate the AKI care bundle.



The first question in the care bundle also recognises that a small proportion of patients who are flagged as AKI on Lorenzo do not need the care bundle completing. These include: patients receiving end of life care, patients in whom the AKI has already resolved, and occasionally, dialysis patients with incorrect results. For these patients, you can select 'No' with the appropriate reason for the first question, 'does the patient need to be on a bundle', and there is no need to complete the other sections.

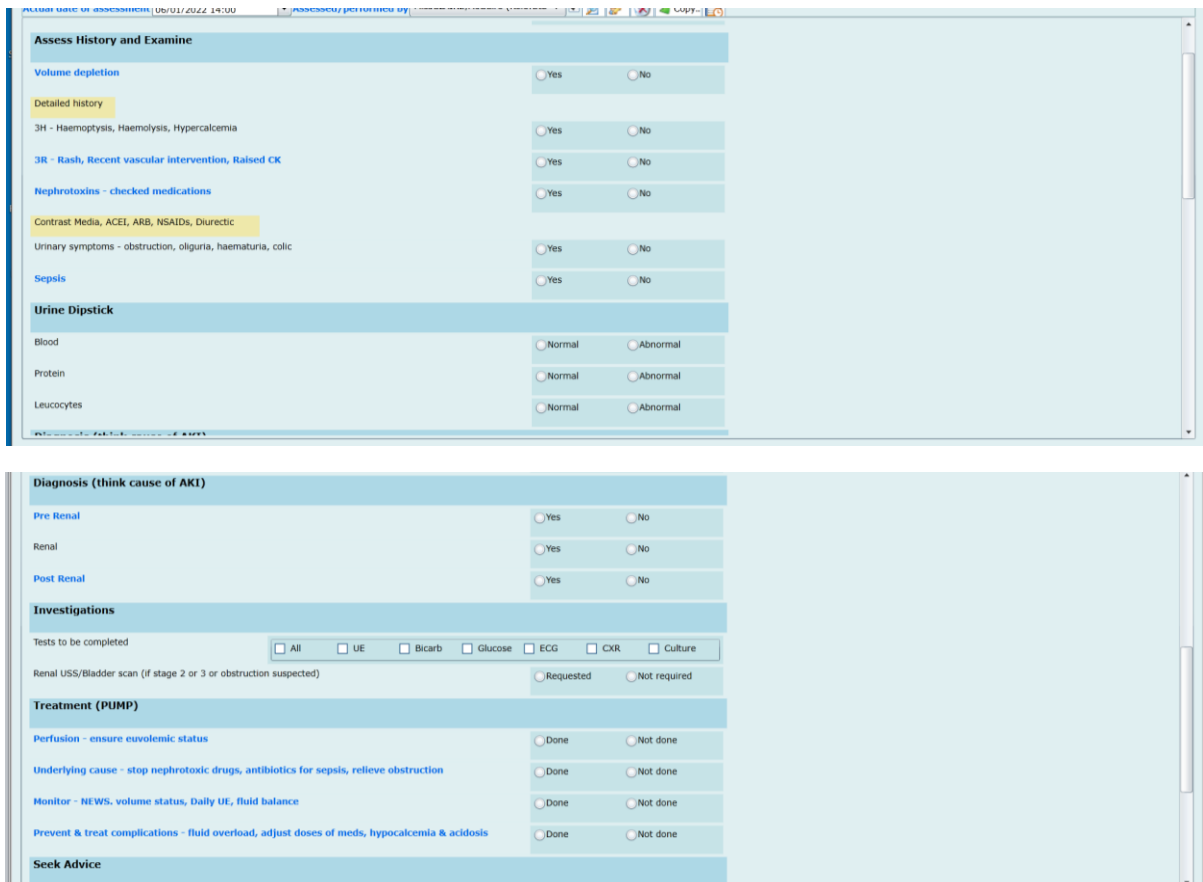


Figure 4 AKI Care Bundle on Lorenzo

Original Note:

AKI Care Bundle
AKI Resolved?: No
 >: If yes, AKI Care Bundle is to be completed and document can be saved

- Assess history & examine
Volume depletion: Yes
 >: Detailed history
3H - Haemoptysis, Haemolysis, Hypercalcemia: No
3R - Rash, Recent vascular intervention, Raised CK: No
Nephrotoxins - Checked medications: Yes
 >: Contrast Media, ACEI, ARB, NSAIDs, Diuretic
Urinary symptoms - Obstruction, oliguria, haematuria, colic: No
Sepsis: Yes

- Diagnosis (Think cause of AKI)
Pre Renal: Yes
Renal: No
Post Renal: No

- Treatment (PUMP)
Perfusion - ensure euvolemic status: Done
Underlying cause - stop nephrotoxic drugs, antibiotics for sepsis, relieve obstruction: Done
Monitor - NEWS, volume status, Daily UE, fluid balance: Done
Prevent & treat complications - fluid overload, adjust doses of meds, hypocalcemia & acidosis: Done

- Seek advice
Seek advice from renal Reg for all AKI stage 3: Done
If specific cause is suspected, refer to Trust AKI website: Done

Summary
 Review Visit
 Notices
 New Results
 Clinical Panels
 Vital Signs
 I & O
 Medications
 Laboratory
 Microbiology
 Blood Bank
 Reports
 Patient Care
 Notes
 Refresh EMR
 Orders
 TTO Review
 Amb Orders
 Clinical Data
 Snapshot
 Plan Of Care
 Worklist
 Mar
 Write Note

Prior Next Amend Undo Unverify Cancel

Figure 5 AKI Care Bundle in Meditech

MANAGEMENT OF AKI

Treat the underlying cause of AKI

Treat co-existing medical problems

Additionally: think PUMP

- **PERFUSION**
 - Volume status. Optimise the volume status, correct relative hypotension, IV fluids when indicated (**further guidance for fluid therapy below**)
 - Withhold BP lowering agents
 - Consider Vasopressors if patient is shocked and not responding to fluids

- **UNDERLYING CAUSE**
 - Sepsis – complete sepsis six care bundle and identify source of sepsis.
 - Medications should be reviewed, looking for those that may potentiate AKI, and dose adjust those that may accumulate –. These are summarised in the traffic light diagram APPENDIX 1 Medicines Optimisation in AKI
 - Obstruction – Catheterise if bladder outflow obstruction is suspected

- **MONITOR**
 - Daily UEs
 - Volume status and fluid balance

- **PREVENT & TREAT COMPLICATIONS** (see COMPLICATIONS OF AKI for more detail)
 - Fluid overload
 - Medication: appropriate dose adjustment (ask pharmacist if unsure)
 - Hyperkalaemia (refer to separate Hyperkalaemia guideline)
 - Metabolic acidosis

INTRAVENOUS FLUID THERAPY

IV fluids should be prescribed as per trust guidelines on Koha

SPECIFIC CONSIDERATION SHOULD BE GIVEN TO THE FOLLOWING;

- Excess fluid associates with worse outcomes in AKI so give if hypovolaemic or hypotensive,
- Don't give fluids just because someone has AKI
- Hartmans contains 5mmol K / L so should be used with caution in AKI

COMPLICATIONS OF AKI

AKI prolongs hospital stay and is associated with increased in hospital and 30 day mortality. Most patients with AKI will however not be looked after or seen by a nephrologist.

The table below lists the complications of AKI that form the basis for **urgent dialysis** decisions and should prompt consideration of referral.

Complications of AKI	Management
Hyperkalaemia	<ul style="list-style-type: none"> ➤ Severe K > 6.5 or if > 5.5 with ECG changes ➤ Refer to Trust Hyperkalaemia Guidelines on koha for detailed guidance <p><i>A copy of the flowchart from these guidelines as attached for reference</i> APPENDIX 2 FLOWCHART FROM HYPERKALEAMIA GUIDELINE</p> <ul style="list-style-type: none"> ➤ Resistant Hyperkalaemia > 7 or ECG changes requires urgent referral for consideration of dialysis
Acidaemia:	<ul style="list-style-type: none"> ➤ pH < 7.25 ➤ Senior Advice should be sought. ➤ Resistant metabolic acidosis due to AKI is an indication for dialysis
Pulmonary oedema	<ul style="list-style-type: none"> ➤ Priority should be to avoid precipitating pulmonary oedema in AKI with accurate fluid assessments. ➤ Dialysis may be indicated if oliguric or diuretic resistance.
Uraemic encephalopathy /pericarditis	<ul style="list-style-type: none"> ➤ This is a rare but serious complication. ➤ Referral to nephrology for consideration of renal replacement therapy should be considered in all cases
Dialysis is useful in a number of drug toxicities (with or without AKI)	<ul style="list-style-type: none"> ➤ Be Guided by Toxbase but early discussion is advised even without AKI

REFERRING TO A NEPHROLOGIST

Management of AKI should be discussed with a nephrologist as soon as possible, and within 24 h of detection **when one or more of the following is present:**

- **Urgent indication for dialysis as per above complications – these patients should be discussed immediately.**
- A possible diagnosis that may necessitate specialist treatment (e.g. vasculitis, glomerulonephritis, tubulo-interstitial nephritis or myeloma).
- AKI with no clear cause.
- AKI that deteriorates despite initial treatment.
- AKI stage 3.
- A renal transplant.
- Chronic kidney disease (CKD) stage 4 or 5.

HOW TO REFER

- **All urgent referrals should be discussed with the Renal SpR on call via RDH switchboard or Bleep 8121 (9 am to 10 PM Monday - Friday, (9am - 9 pm on weekends)** Out of hours the renal consultant on call can be contacted via switchboard.
- Routine Referrals at RDH are via the Renal C2C on ExtraMed and will be picked up the next working day.
- Routine reviews at QHB can be discussed with the Renal SPR covering QHB via switchboard and/or be requested via email to dhft.Renalsecretaries@nhs.net.

AKI Specialist Nurse

- They cover RDH and QHB and, screen and review all appropriate AKI 2 and 3 inpatients. They offer advice and support to the patient and the clinical teams.
- Provide follow-up to all appropriate AKI 2 and 3 patients post discharge, supporting the management of AKI recovery and monitoring kidney function.
- The team are also available to offer telephone advice to clinical teams and deliver AKI education to all grades of staff. Please email them at uhdb.akinurses@nhs.net for further information.

PATIENT RECOVERY AND DISCHARGE PLANNING

- Patients recovering from a significant episode of AKI may develop profound diuresis, resulting in a free water deficit, hypernatremia and/or hypokalaemia. This is especially true if they have had obstructive uropathy that has been relieved.
- Accurate fluid balance with daily weights is very important to prevent patients from becoming dehydrated as they recover from AKI.
- Daily UE should continue into the recovery phase to ensure that recovery is sustained
- If the AKI has not fully resolved on discharge then arrangements need to be made for this to be followed up and medications reviewed, AKI nurses may be able to provide close outpatient followup to prevent a longer hospital stay.

Patients who have had AKI are at **risk of developing CKD in the long term** (this risk depends on the severity of the episode of AKI). This information needs to be communicated to both the patient and the GP. Patients are also at risk **of not restarting medication** after an episode of AKI.

AT DISCHARGE

- Check patient's kidney function prior to discharge
- Refer patients to nephrology if they are discharged with an eGFR <30 mL/min/1.73 m²
- Medications should be reviewed prior to discharge, with a plan to reintroduce medications that may have been held during the acute illness (this may require an early follow-up with the patient's GP)
- The AKI nurses will contact appropriate patients with AKI 2 and 3 to arrange follow-up appointments

DISCHARGE LETTERS TO THE GP SHOULD INCLUDE:

- Severity of AKI
- Cause of AKI
- Kidney function on discharge
- Advice on whether medications need to be reviewed or reintroduced and who is responsible for this
- Any referrals or other follow up arranged

Outpatient Referral

If patients are thought to require ongoing follow up by nephrology then for both Burton and Derby patients please send a referral letter and a copy of the discharge summary with consultant to consultant referral form to renal secretaries. Alternatively complete all details on extramed and tick the box for outpatient referral. Meditech referrals for outpatient follow up will be triaged and seen accordingly.

REFERENCES

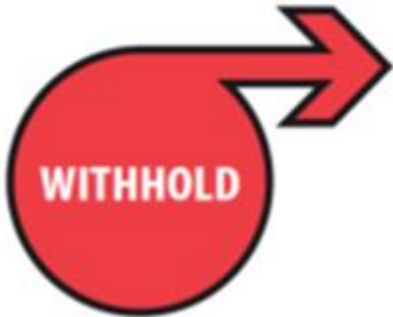
- <https://www.rcplondon.ac.uk/guidelines-policy/acute-care-toolkit-12-acute-kidney-injury-and-intravenous-fluid-therapy>
- <https://www.nice.org.uk/Guidance/CG169>
- <https://www.rcpe.ac.uk/Journal/acute-kidney-injury>
- <http://psnc.org.uk/kentlpc/wp-content/uploads/sites/106/2016/01/AKI-Toolkit.pdf>

Reference Number CG-T/2013/123	Version: 4.1.0		Status Final	
Version / Amendment History	Version	Date	Author	Reason
	4.1.0	Oct 2023	Zoe Pittman	Amendment to appendix (Hyperkalaemia flow chart) as updated in Hyperkalaemia guideline.
Intended Recipients: All Clinicians				
Training and Dissemination: How will you implement the Clinical Guideline, cascade the information and address training				
Development of Guideline: Zoe Pittman Job Title: Consultant Nephrologist				
Consultation with:				
Linked Documents: N/A				
Keywords: Creatinine Estimated Glomerular Filtration Rate (eGFR) Urine Output Hypovolaemia ICM Flowchart Renal Function Renal Injury Renal Insufficiency Acute Kidney Injury (AKI) Dehydration				
Business Unit Sign Off			Group:Renal Date:2023	
Divisional Sign Off			Group: Medicine Division Date:October 2026	
Date of Upload			October 2026	
Review Date			August 2025	
Contact for Review			This should match the author. if different please state who the contact is by Job Title	

APPENDIX 1 Medicines Optimisation in AKI

Medicines Optimisation in Acute Kidney Injury (AKI) and patients at risk of AKI

- Check **Lorenzo** for AKI / AKI alerts / U&E results
- Medication should be reviewed as part of the **AKI care bundle**
- Decisions may vary dependent on the overall clinical picture
- Contact your ward pharmacist or medical team caring for the patient for advice
- Consider withholding medication in patients at risk of AKI
- For dosing see the BNF and consult your ward (or on-call) pharmacist for advice



- WITHHOLD, INFORM DOCTOR, REASSESS**
- NSAIDs (e.g. naproxen, ibuprofen)
 - Diuretics:
Thiazides (e.g. bendroflumethiazide)
K-sparing diuretics (e.g. spironolactone, amiloride)
 - ACE inhibitors & Angiotensin 2 Receptor Blockers (**prijs & sartans**)
 - Metformin
 - Methotrexate
 - Aminosalicylates (e.g. sulfasalazine, mesalazine)
 - Statins & Fibrates
 - Contrast media



- REVIEW DOSE, CONTINUE UNTIL CHECKED WITH DOCTOR**
- Antihypertensives (calcium channel blockers, α -blockers, β -blockers, may need to withhold)
 - Nitrates & nicorandil
 - Opioids (use short-acting preparations, avoid morphine & codeine)
 - Tramadol (avoid long-acting preparations)
 - Benzodiazepines
 - Gabapentin & pregabalin
 - Loop diuretics (e.g. furosemide, bumetanide)
 - Aciclovir
 - Aminoglycosides (e.g. **gentamicin**, amikacin) – nephrotoxic potential, review effective alternatives
 - Penicillins
 - Teicoplanin
 - Trimethoprim (consider alternative as can raise K^+ and Cr)
 - Vancomycin
 - Oral hypoglycaemic agents (e.g. gliclazide, glimepiride, sitagliptin)
 - Levetiracetam
 - Allopurinol, Colchicine



- MONITOR, INFORM DOCTOR**
- **Ciclosporin** (e.g. **Neora[®]**, **Capimune[®]**)
 - **Tacrolimus** (e.g. **Prograf[®]**, **Adoport[®]**, **Advagra[®]**)
 - Digoxin
 - Lithium (e.g. **Priadel[®]**)
 - Phenytoin
 - Warfarin, DOACs (e.g. rivaroxaban)

This document acknowledges the work of Think Kidneys and Bradford Hospitals

APPENDIX 2 FLOWCHART FROM HYPERKALEMIA GUIDELINE

