

Guidelines for the Management of Acute Kidney Injury in Adult Patients and Admission to John Walls Renal unit

1. Introduction

- 1.1 Acute kidney injury (AKI) (previously known as acute renal failure) has a universal definition and staging system in use which allows early detection and management. Classification of AKI is defined into 3 stages by severity known as stage 1, stage 2, and stage 3.
- 1.2 The significance of AKI:
 - a) Sometimes preventable
 - b) Is an independent risk predictor of mortality
 - c) Mortality figures are higher for patients with AKI requiring dialysis than for those with AKI who do not require dialysis
 - d) Patients with AKI have longer ITU and hospital stays when compared to those with normal renal function (including those who do not require dialysis)
 - e) AKI can increase the risk of developing chronic kidney disease (CKD) and worsening of underlying CKD
- 1.3 Some patients with AKI will need to be admitted to units where they can access specialist nephrology support for investigation and/or management of their AKI (e.g. intensive fluid/circulatory management, renal biopsy, specific renal disease management etc.). Patients with quickly reversible AKI due to volume depletion, drug toxicity or sepsis do not necessarily require transfer to be managed by the renal team, but may require support from the renal team through discussion after clinical assessment and judgment by the supervising medical team.
- 1.4 If a referral for a patient with AKI is accepted by the renal team, the patient will be transferred and admitted to the renal unit. On admission they will have a rapid medical review by either a renal registrar or consultant. As part of this review process, the need for level 2 care (on Ward 30 HDU) will be assessed.
- 1.5 The critical care referral process should be followed for patients deemed suitable for and requiring escalation to level 3 care. The critical care team should be involved in a timely manner dependent on the patient's clinical condition to facilitate discussions and further management planning.
- 1.6 In 2019 the National Institute for Clinical Excellence (NICE) published Guidelines on the Prevention, Detection and Management of AKI, which have been incorporated into these guidelines where appropriate.

2. Scope

- 2.1 This guideline is for use by all medical, nursing, pharmacy and dietetic staff involved in looking after **adult** patients who are admitted with or acquire AKI during their hospital stay.
- 2.2 These guidelines are not designed to be used in obstetric patients with pre-eclampsia, please see Guidelines for the Management of severe pre-eclampsia / eclampsia Trust Ref: C3/2001

2.3 This guideline is designed to assist medical staff to assess and identify those appropriate to transfer to nephrology.

2.4 It is also designed to assist medical and nursing staff in the nephrology department to transfer safely and quickly patients with AKI from other wards or hospital sites to the nephrology wards.

3. Guideline Statements

The UHL AKI Alert Sticker and Care Bundle are to be used by clinical staff involved in looking after adult patients who present with AKI and can be found in Appendix I and II.

Algorithms for transfer from different locations/hospital sites can be found in Appendix III.

This section provides further guidelines and information on the following:

3.1. Definition of AKI	3.10 Specific Drug Therapies
3.2. Diagnosis and Staging of Acute Kidney Injury	3.11. Nephrology advice
3.3. Identifying Patients at Risk of AKI	3.12. Indications for kidney replacement therapy.
3.4. Preventing 'At Risk' Patients from Developing AKI	3.13. Careful consideration for kidney replacement therapy
3.5. Contrast Agent: Risks and Administration	3.14. AKI transfer to renal unit safety criteria
3.6. Assessment of the Patient with AKI	3.15. Dietician referral
3.7. Determination of likely cause of AKI	3.16. Daily review
3.8. Investigation of the causes of AKI	3.17. Information and Support for Patients and Carers
3.9 General Management of AKI	3.18. Discharge and Follow up for Patients with AKI

3.1	<p><u>Definition of AKI</u></p> <p>Clinically AKI is characterised by a rapid reduction in kidney function (over hours or days) resulting in a failure to maintain fluid, electrolyte and acid-base homeostasis</p> <p>AKI is defined as any of the following:</p> <ul style="list-style-type: none"> • Increase in SCr by ≥ 26.5 micromol/L within 48 hours; OR • Increase in SCr to ≥ 1.5 times baseline, which is known or presumed to have occurred within prior 7 days; OR • Urine volume <0.5 ml/kg/hr for 6 hours
3.2	<p><u>Diagnosis and Staging of Acute Kidney Injury</u></p> <p>a) Diagnosis and staging of AKI is based on serum creatinine concentration (SCr) and/or urine output.</p> <p>b) Using the NHS England AKI detection algorithm, an AKI Alert is generated via ICE/iLab/Nervecentre for every creatinine result that is consistent with AKI informing the clinician of the patient's stage of AKI.</p> <p>c) Be mindful that the AKI alert may be automatically triggered inappropriately in those who are on dialysis already (Haemodialysis or Peritoneal dialysis), and it is not possible for a patient who has End-Stage Kidney Disease on maintenance renal replacement therapy to have AKI.</p> <p>(c) Estimated glomerular filtration rate (eGFR) is not used in the assessment of patients with AKI. The GFR is only valid when serum creatinine is in a steady state.</p> <p>*Must have met initial criteria for definition of AKI</p>

AKI Stage	Serum Creatinine Criteria	Urine Output Criteria
1	Rise in SCr \geq 26 micromol/L or SCr \geq 1.5 - 1.9 x the baseline	<0.5 mL/kg/hr for >6 hrs
2	Rise in SCr > 2 - 2.9 x the baseline	<0.5 mL/kg/hr for >12 hrs
3	Rise in SCr >3 x the baseline or *SCr \geq 354 micromol/L or initiated on RRT (irrespective of stage at time of initiation)	<0.3 mL/kg/hr for 24 hrs or anuria for 12 hrs KDIGO Staging System (2012)

3.3	<p>Identifying Patients at Risk of AKI</p> <p>Specific co-morbidities associated with AKI are:</p> <ol style="list-style-type: none"> Chronic kidney disease (CKD) Sepsis Cardiac failure Liver disease Diabetes mellitus Nephrotoxic medication or on ACE-Inhibitors or angiotensin-II receptor blockers especially if hypovolaemic Age > 65years Hypertension or vascular disease Post-operative hypotension History of AKI Had Contrast Agent administered within the last 7days
3.5	<p>Contrast Agent risks and Administration</p> <p>may mean limited access to fluids due to reliance on central venous access.</p> <p>Please refer to the IV contrast guideline for considerations about renal disease (currently under revision) – Radiology Contrast SOP.es</p> <p>Monitor serum creatinine at clinically appropriate intervals in all patients who are at risk of AKI</p>
3.6	<p>Risks of AKI of the Patient with AKI</p>
3.4	<p>Preventing Patients from Developing AKI</p> <p>Preventing Patients from Developing AKI</p> <ol style="list-style-type: none"> Avoid potentially nephrotoxic drugs in patients with pre-existing renal, intrinsic renal or obstructive AKI. Maintain adequate blood pressure If volume depleted with no evidence of cardiac failure, infuse IV isotonic crystalloid. Assessment of the patient's volume status must be carefully and continuously monitored during IV fluid therapy. <ul style="list-style-type: none"> Systemic symptoms (fatigue, fever, weight loss, epistaxis, haemoptysis, joint pain) Difficulty passing urine Reduced urine output Avoid any nephrotoxins where possible (consider the impact on other diseases being treated) e.g. NSAIDs and antibiotics (gentamicin, amikacin), amphotericin and intravenous or intrathecal iodinated contrast media. If no alternatives exist, use the appropriate 'renal' doses. Record the patient's drug history. NOTE Refer to WHU's microbiology website for dosing of gentamicin and vancomycin in patients with CKD (go and hit always by teardrop) with the drug history). Book for the patients developing rigidity of the joints which are treated by using the Sepsis Care Bundle (Trust Ref: B11/2014)

	<p>Physical Examination</p> <p>a) Assess the patient's volume status. This should include checking the patient's:</p> <ul style="list-style-type: none"> • BP with comparison to the patient's normal BP and postural drop • Heart rate with postural change • JVP or rarely CVP if volume assessment is difficult • Lung bases for evidence of fluid • Peripheries and sacrum for oedema • Weight • Fluid balance (input and output charts) <p>b) Full examination of CVS, Respiratory System and Abdominal Examination</p> <p>c) Look for evidence of skin rashes</p> <p>d) Male patients who may have obstructive cause should have PR examination to assess prostate</p> <p>e) Consider pelvic examination in female patients who present with obstruction</p>
<p>3.7</p>	<p><u>Determination of likely cause of AKI</u></p> <p>AKI in a hospital setting is often <u>multifactorial</u>. It is still useful to consider AKI in terms of:</p> <p>a) Pre-renal AKI</p> <p>This is a decreased glomerular filtration rate resulting from renal hypo-perfusion. In pre-renal failure, the kidney remains structurally normal and therefore the condition is rapidly reversible when the underlying cause is corrected. Renal autoregulation generally becomes impaired when mean arterial pressure fall below 70 mmHg. Drugs which interfere with renal autoregulatory processes (NSAIDs, ACE-Inhibitors and Angiotensin II Receptor blockers) as well as excessive use of loop diuretics can also increase risk of AKI.</p> <p>b) Intrinsic AKI</p> <p>The commonest cause of intrinsic AKI is acute tubular necrosis (ATN) due to any of the causes of pre-renal AKI (if not reversed before tissue damage occurs), tubular nephrotoxins (such as aminoglycosides and myoglobin) or sepsis. Other causes include glomerulonephritis, interstitial nephritis and vascular causes.</p> <p>c) Post-renal AKI</p> <p>This is less common than the other two categories but is important to consider as prompt diagnosis and intervention may result in full recovery. <u>Relief of the obstruction often leads to a significant diuresis which requires careful management of fluid balance to prevent the subsequent development of pre-renal AKI.</u></p> <p>d) Acute on Chronic Kidney Disease</p> <p>It is important to try and establish whether the patient has underlying renal impairment as it may save unnecessary investigation and the approach to management may be different. This can often be established by looking for previous measures of kidney function on the ICE pathology system.</p> <p>75% of AKI is due to either pre-renal failure or acute tubular necrosis (ATN) and quick diagnosis and treatment may prevent the need for dialysis and the associated increase in morbidity and mortality that results.</p>

e) Consider the following when investigating the potential causes of AKI

- Volume depletion
- Sepsis
- Medications which could be injurious to the kidney
- Obstruction
- Contrast Agent
- Intrinsic Renal Disease
- Rhabdomyolysis

UHL have adopted the use of an AKI Alert sticker (Appendix I) to promote early recognition of potential causes of AKI and encourage timely management using the AKI Care Bundle Actions (Appendix II). (These can be ordered from the Print room ref no: AKI01)

3.8

Investigation of the causes of AKI

In order to aid detection of the cause(s) of AKI all patients with AKI should have the following:

a) URINE DIPSTICK ANALYSIS

- Perform urine dipstick testing for blood, protein, leucocytes, nitrites and glucose in all patients as soon as AKI is suspected or detected. Document the results on Nervecentre, and ensure that appropriate action is taken when results are abnormal.
- Think about a diagnosis of acute nephritis and referral to the nephrology team when a patient with no obvious cause of AKI and has urine dipstick results showing haematuria and proteinuria, without urinary tract infection or trauma due to catheterisation. Send urine for MSU, urine Protein:Creatinine Ratio and Albumin:Creatinine Ratio (PCR & ACR).

b) Blood Tests

- Biochemistry screen (renal profile which also includes bicarbonate and chloride, bone biochemistry, LFTs, CRP, glucose)
- FBC
- Clotting screen
- Blood cultures (if evidence of sepsis)

c) Other Investigations

- ECG
- CXR

d) Review of Nephrotoxic Medications

Review the patient's recent drug history for any nephrotoxins including over the counter and herbal medicines. This includes any contrast agent administered within the last 7 days.

e) USS renal tract

Do not routinely offer an ultrasound of the urinary tract when a plausible cause of AKI has been identified.

A renal ultrasound is useful to look for signs of obstruction and renal size (small scarred kidneys signify CKD; asymmetric kidneys suggest renal artery stenosis or a non-functioning kidney).

- USS should be performed within 24 hours of assessment for all patients suspected of urinary tract obstruction and for those with AKI stage 2 or 3 with AKI of unknown cause. If the patient is obstructed contact should be made IMMEDIATELY with the urologists or interventional radiologists.
- It should be considered for those with acute on chronic renal impairment.
- If pyonephrosis (infected & obstructed kidney(s)) is suspected in patient with AKI immediate ultrasound should be carried out (within 6hrs of assessment).

f) Where the history is suggestive, patients should also be tested for:

- Creatinine Kinase (CK) if rhabdomyolysis suspected
- Renal immunology (ANCA, anti-GBM, ANA, dsDNA, RhF, C3 and C4) in patients with suspected connective tissue disease or vasculitis including all patients with AKI who have blood and protein in the urine
- Serum electrophoresis/immunoglobulins and serum free light chains in all patients who may have myeloma (elderly, anaemia or hypercalcaemia)
- Anti-streptolysin O (ASOT) if patient likely to have had recent streptococcal infection.
- Blood film and LDH for all patients with a low Hb and low platelet count to check for haemolysis and microangiopathy – please contact renal SpR on-call if suspected thrombotic microangiopathy
- Arterial blood gases for acid-base balance or gas exchange if any evidence of respiratory or cardiovascular compromise or low venous bicarbonate

3.9 General Management of AKI

The UHL AKI Care Bundle can be found in Appendix II

a) Monitoring patients with AKI

The following should be recorded for all patients with AKI:

- Pulse rate, BP, respiratory rate and O₂ saturation (At least 4 hourly NEWS irrespective of score or more frequently if NEWS pathway advises)
- Hourly urine output in patients with urinary catheter
- Strict input and output recorded on fluid balance charts including losses from GI tract, drains etc.
- Daily blood tests – renal profile, bone and venous bicarbonate
- Daily weight
- Ensure adequate nutrition

b) Treatment

General treatment-refer to the AKI Care Bundle (Appendix II). Priorities in treating AKI are:

- Early identification of underlying & remediable causes
- Assess fluid status and optimise fluid balance – Consider catheterisation.
- Identify patients who require renal replacement therapy (RRT) and start treatment at the appropriate time.
- Review of medication and dosage
- Early referral to the nephrologist where appropriate

Daily review by the medical team responsible for the patient is imperative.

c) Assess Fluid Status

Volume depletion is a common cause of AKI however AKI can also occur in the context of volume overload and a thorough examination of the patient is mandatory.

- a) If the patient is obviously volume deplete then intravenous fluids should be initiated without the immediate need for CVP monitoring. Fluid status should be regularly reassessed.
- b) Caution should be taken when administering IV fluids especially in patients at risk of fluid overload (the elderly, known heart failure or CKD). If in doubt seek senior advice. Small boluses of IV fluids with frequent re-assessment of fluid status may be the safest approach.

	<p>c) If there is doubt about a patient's fluid balance then CVP monitoring may be considered as an aid to assess fluid status. This should only be carried out following discussion with the Senior Clinician and Critical Care Outreach/ Critical Care.</p> <p>d) <u>It is imperative that fluid status is regularly reassessed and that a strict record of input and output is maintained.</u></p> <p>d) Pharmacological Management</p> <p>a) Review medication for any nephrotoxic drugs i.e. NSAIDs, gentamicin</p> <p>b) Consider <u>temporarily</u> withholding drugs which interfere with renal haemodynamic (e.g. ACEi, ARBs, SGLT2i) – this should be done following consideration of cardiovascular indications and discussion with a senior clinician and pharmacist</p> <p>c) Drug doses need to be adjusted appropriately in all patients with AKI. Advice should be sought from pharmacy or microbiology in the first instance.</p> <p>d) Drug levels should be monitored for drugs with a narrow therapeutic index which are excreted by the kidney (e.g. digoxin, vancomycin, and gentamicin).</p> <p>e) Avoid combining fluids & diuretics</p> <p>f) If nephrotoxic medication withheld this must be reviewed regularly and the GP notified of any changes and follow up required.</p>
<p>3.10</p>	<p><u>Specific Drug Therapies</u></p> <p>To date there are no pharmacological interventions which have been shown to prevent or treat AKI. Furosemide and sodium bicarbonate may be used cautiously in specific circumstances.</p> <p>a) Furosemide</p> <p>Furosemide is often withheld in the context of AKI. In many cases (such as dehydration/sepsis), this is appropriate. However, it is important to note that in certain circumstances, diuretic therapy may be indicated, (e.g. in patients with decompensated heart failure, congestion is the primary driver for AKI and decongestion with diuretics is the key to reducing symptoms and improving patient outcomes.) It is therefore important to carefully assess the patient's fluid volume status and if the patient is overloaded, Furosemide may be indicated.</p> <p>Furosemide can be used for a short time to manage volume overload in people awaiting initiation of renal replacement therapy (RRT). However meta-analysis has shown that furosemide gave no benefit in terms of overall mortality, the need for RRT or the duration of RRT. Furthermore high doses can be associated with an increased risk of ototoxicity. Its use should not delay referral to nephrology or initiation of RRT.</p> <p>Furosemide in cases of hyperkalaemia or hypercalcaemia:</p> <p>Removal of potassium and calcium both rely on the delivery of salt and volume to the distal nephron. In these circumstances, furosemide can aid urinary removal of potassium and calcium and is a useful adjunct to intravenous fluids in patients where rapid reduction in serum levels is required or in whom fluid overload is a risk.</p> <p>It is obviously not useful in patients with oliguria/anuria, in these circumstances dialysis is usually required to correct the electrolyte disturbance.</p> <p>b) Sodium Bicarbonate</p> <p>Isotonic sodium bicarbonate (1.4% or 1.26%) is sometimes warranted in the treatment of AKI. However it results in a large sodium load and the production of carbon dioxide (CO₂). It should therefore not be given to patients who are already fluid overloaded and should be used with extreme caution in patients at risk of fluid overload (elderly, heart failure or existing CKD) unless obviously volume deplete.</p> <p>It should also not be used in patients with type 2 respiratory failure or at risk of respiratory compromise who cannot 'blow off' the excess CO₂. Nephrology advice should be sought before administration if there is uncertainty about whether sodium bicarbonate should be used in an individual patient.</p>

	<p>c) Dopamine</p> <p>Dopamine should NOT be used to treat AKI. There is no evidence for the use of dopamine in any circumstances in AKI and its use can lead to cardiac arrhythmias and myocardial and intestinal ischaemia.</p> <p>d) N-acetylcysteine</p> <p>Several randomised studies have shown that N-acetylcysteine is ineffective at preventing AKI.</p>
3.11	<p>Nephrology Advice</p> <p>The following patients should be discussed with the on-call Nephrology SpR (send referral via www.referapatient.org to Renal Medicine at Leicester General Hospital)</p> <ul style="list-style-type: none"> a) Patients with Stage 3 AKI b) Patients with possible diagnosis that may need specialist treatment (e.g. vasculitis, glomerulonephritis, tubulointerstitial nephritis or myeloma) c) Patients with an unexplained cause of AKI (regardless of stage) d) Patients with inadequate response to treatment e) Patients who have had a renal transplant f) Patients with CKD 4 or 5 g) Patients with complications associated with AKI <p>Those needing advanced respiratory support or basic respiratory support plus support of two or more other organs should receive level 3 (intensive, ITU) care.</p>
3.12	<p>Indications for Kidney Replacement Therapy</p> <p>!! Patients who require urgent referral for renal replacement therapy !!</p> <ul style="list-style-type: none"> a) Hyperkalaemia unresponsive to medical treatment b) Fluid overload unresponsive to medical treatment c) Persistent or worsening metabolic acidosis d) Uraemic symptoms (intractable vomiting, confusion, twitching) or evidence of pericardial effusion
3.13.	<p>Careful Consideration for Renal Replacement Therapy</p> <p>Before referring to the renal team, it is important to consider whether the patient would be suitable for renal replacement therapy (RRT). RRT for AKI is usually in the form of haemodialysis. There are well-known risks associated with the haemodialysis procedure, including hypotension, arrhythmia, membrane bio-incompatibility, and complications of vascular access and anticoagulant administration. There is also some concern that RRT may compromise recovery of renal function and increase the progression of CKD (KDIGO guidelines AKI).</p> <p>RRT for patients with AKI, in most cases is temporary, but can be permanent if kidney function does not recover. In these cases, it is important to consider whether haemodialysis will add a significant burden to a patient without adding much benefit to their quality of life. Factors that are associated with an increased risk of mortality include age > 70 years, presence of previous chronic illness/co-morbidities, AKI caused by cardiac failure and infection and increasing number of failed organs.</p> <p>It is important to consider a holistic approach and remember that dialysis will clear the toxins building up from AKI, but will not help with any ongoing other medical issues including, other organ dysfunction, physiological reserve, functional status/frailty.</p> <p>For those who are haemodynamically unstable, CRRT in ICU may be better tolerated than intermittent haemodialysis.</p>

3.14 **AKI Transfer to Renal unit safety criteria**

Patients referred for transfer to the renal unit should have been assessed by an experienced doctor defined as an ST4 or above. The responsibility for ensuring the patient is safe to transfer rests with the referring team.

All patients transferring to the renal unit (regardless of where they are transferring from) should meet the following safety criteria (adapted from London AKI Network Manual 2015) :-

1. Hyperkalaemia

- No ECG changes
- $K < 6.5$ *
- If K lowered to <6.5 after presentation this must be sustained e.g. bicarbonate therapy or sodium zirconium cyclosilicate (Lokelma) not transient therapy (insulin and dextrose).

*If $K^+ > 6.5$ mmol/L and transfer to renal unit still deemed the most appropriate action, this must be discussed and agreed with the nephrology consultant on-call prior to any transfer

2. Renal Acidosis

- pH >7.2
- Bic >12 mmol/L
- Lactate < 4 mmol/L
- Respiratory rate < 24 /min

(N.B Renal acidosis does not have the same prognostic implications as acidosis due to hypo-perfusion)

3. Circulatory

- HR <120 /min
- BP >100 mmHg systolic
- MAP >65 mmHg
- Lactate <4 mmol/L

(lower BP values may be accepted if it has been established clearly these are 'normal' pre-morbid)

4. Respiratory

- Respiratory rate < 24 /min
- Oxygen saturation $>94\%$ and not more than 35% oxygen
 - If patient has required acute CPAP, must have been independent of this treatment for 24 hours

5. Neurological

- GCS >12 or alert on AVPU scale

Patients not fulfilling criteria for safe transfer

Patients who do not fulfil criteria for safe transfer should be referred to local ICU for assessment, admission or stabilisation.

Once stabilised, follow ICU to renal unit transfer policy (Appendix III).

3.15	<p>Dietician referral</p> <p>Patients with AKI often have protein-energy wasting and their nutritional requirements vary considerably depending on the course of the AKI and the nature of their underlying illness. Poor nutrition has been shown to be a negative prognostic factor in AKI.</p> <p>Consider dietetic referral in all patients with AKI and refer all who fulfil the NICE criteria for nutritional support.</p>
3.16	<p>Daily review</p> <p>Review of the patient and their blood tests by the medical team responsible for their care, <u>must</u> occur on an at least daily basis regardless of which ward the patient may move to. These patients must be handed over between shifts with up-to-date information about which ward they are on to ensure assessment occurs during weekends/bank holidays etc.</p>
3.17	<p>Information and Support for Patients and Carers</p> <p>It is important to keep patients informed of their diagnosis of Acute Kidney Injury and to discuss immediate treatment options, monitoring and prognosis. A UHL Patient Information Leaflet is available to provide information and guidance on self-management and support and should be given to patients with AKI and/or their carers (This can be ordered from the Print room, Reference Code: AKI02)</p>
3.18	<p>Discharge and Follow up for Patients with AKI</p> <p>Patients who have had an episode of AKI must have this recorded in their discharge summary including the worst stage of AKI during admission and the stage of AKI on discharge. The GP should be offered advice about repeat testing of kidney function and specific guidance about restarting any drugs which may have been temporarily withheld (e.g. ACEi, ARBs).</p> <p>Formal post-discharge nephrology review should be arranged:</p> <ul style="list-style-type: none"> • Within 90 days for those with residual CKD stage G4 at hospital discharge • Within 30 days for those with residual CKD stage G5 (non-dialysis-requiring) at hospital discharge • Within 30 days for those with ongoing dialysis requirements at the time of hospital discharge

4 Education and Training

Education is key to improving AKI outcomes. An AKI e-learning module is available for all clinical staff and can be accessed via eUHL.

5 Monitoring and Audit Criteria

Element to be Monitored	Lead	Method	Frequency	Reporting arrangements
Review of patients with “new hospital acquired” AKI	Dr Apexa Kuverji	Deteriorating Patient Board report	Every quarter/year	AKI T&F Group
Management of patients with AKI	Dr Apexa Kuverji	Case note and Nervecentre audit	Annually	AKI T&F Group
Communication of AKI to Primary Care	Dr Apexa Kuverji	Audit of Discharge Letter	Annually	AKI T&F Group

6 Legal Liability Guideline Statement

Guidelines or Procedures issued and approved by the Trust are considered to represent best practice. Staff may only exceptionally depart from any relevant Trust guidelines or Procedures and always only providing that such departure is confined to the specific needs of individual circumstances. In healthcare delivery such departure shall only be undertaken where, in the judgement of the responsible healthcare professional it is fully appropriate and justifiable - such decision to be fully recorded in the patient's notes.

7 **Supporting Documents and Key References**

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8. **Key Words**

Acute Kidney Injury, AKI, Staging, Care Bundle

DEVELOPMENT AND APPROVAL RECORD FOR THIS DOCUMENT	
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Version Number: 4	Details of Changes made during review: V4 Reviewed by the Deteriorating Patient Board 21/08/2023 <ul style="list-style-type: none"> • Guidelines for referral to John Walls Renal Unit merged with AKI guidelines • Added section on careful consideration of renal replacement therapy • Added comment on AKI alerts in ESKD patients • Added Lokelma as a management option for hyperkalaemia • Contrast induced nephropathy section removed and signposted to separate guideline. • More detail added regarding use of Furosemide in cardiorenal AKI • Referral process via www.referapatient.org updated • Information regarding discharge and follow up updated according to NICE guidelines • AKI referral criteria added as Appendix III
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Appendix I – AKI Alert Sticker

Below is an example of the AKI Alert Sticker – approved version is available from the print room – order reference AKI01

ACUTE KIDNEY INJURY ALERT REVIEW STICKER					
Patient's Name:				Hospital No:	
Date:	Time:	Creatinine:	AKI STAGE:	Signature:	
Investigate potential causes	Present?	Treatment/ Comments	Care Bundle Actions	Done Or N/A	Comments
Volume depletion		If being treated for Heart Failure do not give fluid until reviewed by Senior Clinician	<ul style="list-style-type: none"> Assess volume state & monitor fluid balance. Review regularly Avoid combining fluids and diuretics Perform urinalysis – if blood/protein positive send for MSU 		
Sepsis					
Nephrotoxic Medication			Review nephrotoxic medication (ACEi / ARB / diuretics / NSAID / antibiotics)		
Urinary tract obstruction			Order renal ultrasound if urinary tract obstruction suspected or cause of AKI Stage 2 or 3 is unclear.		
Contrast agent within last 7 days			Discuss with Nephrology if <ul style="list-style-type: none"> Intrinsic renal disease/vasculitis suspected (i.e. deteriorating AKI with blood++ and/or protein++) Renal function deteriorating Patient has renal transplant 		
Intrinsic renal disease			Consider appropriateness of renal replacement therapy if hyperkalaemic / acidotic / pulmonary oedema not responding to diuretics		
Rhabdomyolysis			Inform patient of diagnosis and likely cause and give Information Leaflet		
COMMUNICATING WITH PRIMARY CARE Discharge Letters should include: AKI Stage, medication review details, type and frequency of post-discharge blood tests and future plan					

This Care Bundle should be used in conjunction with the Management of Acute Kidney Injury (AKI) In Adult Patients Guidelines (Trust reference B21/2009) and is for use by all clinical staff involved in looking after adult patients who present with AKI excluding obstetric patients with pre-eclampsia.

AKI Stage 1

Rise in SCr > 1.5 - 1.9 x the baseline
 or >26 micromol/L within 48hrs
 Or
 U/o < 0.5 ml/kg/hr for > 6hrs

AKI Stage 2

Rise in SCr of >2 – 2.9 x the
 baseline
 Or
 u/o < 0.5ml/kg/hr for

AKI Stage 3

Rise in SCr of 3 x baseline to >354 micromol/L
 Or
 u/o < 0.3 ml/kg/hr for ≥ 24hrs
 or anuria for ≥ 12hrs

Investigate Potential Causes of All Stages of AKI

- **Volume status**– Is there evidence of (a) reduced circulating volume **OR** overload and renal hypoperfusion because of cardiac failure? This distinction is critical – fluids are **NOT** appropriate in the context of heart failure until discussed with a senior doctor.
- **Sepsis** - is there known infection or suspicion of infection?
- **Nephrotoxic medication**– Have any drugs been started recently? Are these known to be nephrotoxic? Could they cause tubulo-interstitial nephritis?
- **Has Contrast Agent been** administered within the last 7 days?
- **Exclude renal tract obstruction**

If the cause is unclear consider:

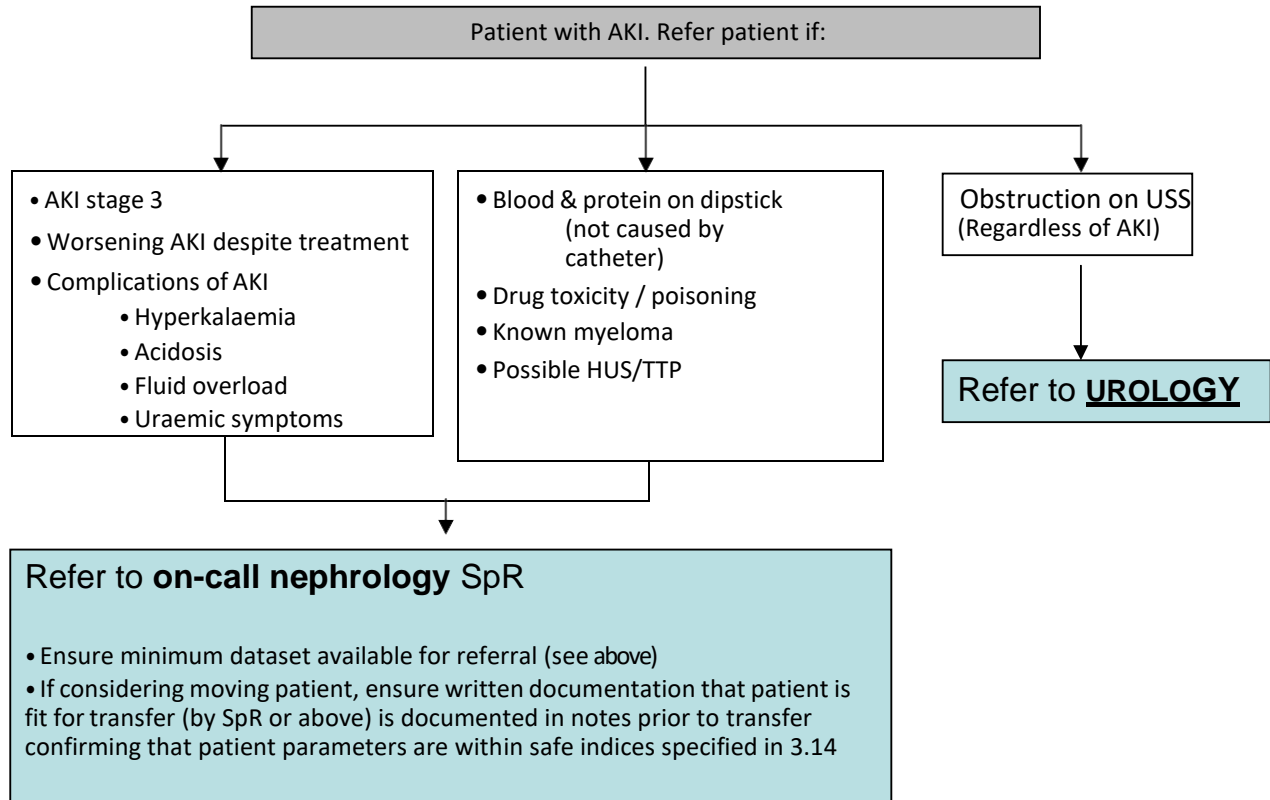
- **Intrinsic renal disease** – deteriorating renal function with urine blood++ and/or protein++
- **Rhabdomyolysis**
- **Thrombotic microangiopathies**
- **Occult infection**
- **Hepatorenal syndrome**

AKI Care Bundle

- **Assess volume status:** Is the patient volume depleted or overloaded? If volume depleted give bolus fluids until volume replete with regular review of response. If volume overloaded, consider diuretics – IV fluids should not be given except after senior review. **Avoid combining fluids and diuretics.**
- **Perform Urinalysis:** If blood +/- protein ++ present check MSU and urine PCR
- **If cause unclear and vasculitis suspected** – check ANA, ANCA, GBM, RhF, hepatitis B and C serology. Include venous bicarbonate, creatinine kinase, venous blood cultures, liver function tests, C- RP with initial blood tests. Consider blood film, LDH and haemolysis screen if Hb and/or platelets are low.
- **Consider bladder catheterisation**
- **Monitor Fluid Balance & daily weights.** Perform regular fluid assessments & check for signs of uraemia
- **EWS Observations-** at least 4hrly
- **Consider (consequences of) stopping or dose adjusting nephrotoxic medication** (e.g. NSAID /metformin/ gentamycin/ ACE inhibitors/ ARB /diuretics.). **Review all drug doses.**
- **Measure U&Es, bone and venous bicarbonate daily whilst creatinine continues to climb.**
- **Order Renal Ultrasound:** Urgently (within 24hrs of assessment) if urinary tract obstruction suspected or 6hrs if obstruction with infection suspected (pyonephrosis) or if no cause identified for AKI Stage 2 or 3
- **Discuss with Nephrology if**
 - Renal disease suspected (i.e. deteriorating AKI with haematuria +/- proteinuria)
 - Inadequate response to treatment
 - Patient has renal transplant.
 - Patient requires renal replacement therapy (increasing uraemia/ hyperkalaemic/ acidotic/ pulmonary oedema not responding to diuretics)

Appendix III

AKI referrals from UHL wards



AKI Transfer policy from UHL ICU to renal unit

Patient on ICU deemed fit for transfer by ICU consultant

- Phone renal SpR to review patient on ICU (GGH only) or if not at GGH, renal SpR on-call to get verbal handover from ICU SpR then arrange transfer to renal wards; generally this will be to renal HDU
- Aim to transfer within 24 hours of being deemed fit to step down from ITU otherwise if delay, repeat assessment as above is required.
- Below is consensus guideline for safe transfer from ICU to renal wards (taken from North London guidelines):-

Metabolic

K < 6.0, ionised Ca > 1 pH

normal Bicarbonate > 16

Lactate normal

Respiratory Respiratory rate

< 24

Saturations > 94% on not more than 35% oxygen

If patient required acute CPAP patient must have been independent of this treatment for 24 hours

If ventilated < 1 week patient should have been independent of respiratory support for 48 hours

If longer term invasive ventilation patient should have been independent of all respiratory support for

1 day for each week ventilated and for a period not less than 48 hours.

Circulatory HR <

120

BP > 100mmHg systolic MAP >

65MMHg

Lactate < 4