

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 Acute Kidney Injury:
 - a) Sometimes preventable,^{2,3}
 - 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 In 2013 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 acute kidney injury 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

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 One and Two.

This section provides further guidelines and information on the following:

3.1. Definition of AKI	3.9. General Management of AKI
3.2. Diagnosis and Staging of Acute Kidney Injury	3.10. Specific Drug Therapies
3.3. Identifying Patients at Risk of AKI	3.11. Nephrology Advice
3.4. Preventing 'At Risk' Patients from Developing AKI	3.12. Dietician referral
3.5. Contrast Agent: Risks and Administration	3.13. Daily review
3.6. Assessment of the Patient with AKI	3.14. Information and Support for Patients and Carers
3.7. Determination of likely cause of AKI	3.15. Discharge and Follow up for Patients with AKI
3.8. Investigation of the causes of 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 homoeostasis.¹</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 algorithm⁵, an AKI Alert is generated via ICE and iLab for every creatinine result that is consistent with AKI informing the clinician of the patient's stage of 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 AKIA</p> <table border="1" style="width: 100%; border-collapse: collapse;"> <thead> <tr> <th style="background-color: #e0e0e0;">AKI Stage</th> <th style="background-color: #e0e0e0;">Serum Creatinine Criteria</th> <th style="background-color: #e0e0e0;">Urine Output Criteria</th> </tr> </thead> <tbody> <tr> <td style="background-color: #00ffff; text-align: center;">1</td> <td>Rise in SCr ≥ 26 micromol/L or SCr $\geq 1.5 - 1.9$ x the baseline</td> <td><0.5 mL/kg/hr for >6 hrs</td> </tr> <tr> <td style="background-color: #00bfff; text-align: center;">2</td> <td>Rise in SCr $> 2 - 2.9$ x the baseline</td> <td><0.5 mL/kg/hr for >12 hrs</td> </tr> <tr> <td style="background-color: #0099ff; text-align: center;">3</td> <td>Rise in SCr >3 x the baseline or *SCr ≥ 354 micromol/L or initiated on RRT (irrespective of stage at time of initiation)</td> <td><0.3 mL/kg/hr for 24 hrs or anuria for 12 hrs</td> </tr> </tbody> </table> <p style="text-align: right; font-size: small;">KDIGO Staging System (2012)</p>			AKI Stage	Serum Creatinine Criteria	Urine Output Criteria	1	Rise in SCr ≥ 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 ≥ 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
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<p>3.3</p>	<p>Identifying Patients at Risk of AKI</p> <p>Specific co-morbidities associated with AKI are:</p> <ul style="list-style-type: none"> a) Chronic kidney disease (CKD) b) Sepsis c) Cardiac failure d) Liver disease e) Diabetes mellitus f) Nephrotoxic medication or on ACE-Inhibitors or angiotensin-II receptor blockers especially if hypovolaemic g) Age > 65years h) Hypertension or vascular disease i) Post-operative hypotension j) History of AKI k) Had Contrast Agent administered within the last 7days <p>Monitor serum creatinine at clinically appropriate intervals in all patients who are at risk of AKI</p>
<p>3.4</p>	<p><u>Preventing Patients from Developing AKI</u></p> <ul style="list-style-type: none"> a) Avoid pre- and peri-operative hypovolaemia. b) Maintain adequate blood pressure c) If volume deplete with no evidence of cardiac failure, infuse i.v. isotonic crystalloid. <u>Assessment of the patient's volume status must be carefully and continuously monitored during i.v. fluid therapy.</u> d) Avoid any nephrotoxins where possible (consider the impact on other diseases being treated) e.g. NSAIDs, aminoglycosides (gentamicin, amikacin), amphotericin and intravenous or intra-arterial iodinated contrast media. If no alternatives exist, use the appropriate 'renal' doses. <p>NOTE: refer to UHL's microbiology website for dosing of gentamicin and vancomycin in patients with CKD (go to UHL microbiology website)</p> <ul style="list-style-type: none"> e) Be alert to the patient developing signs of sepsis and treat promptly using the Sepsis Care Bundle (Trust Ref: B11/2014)

<p>3.5</p>	<p><u>Contrast Agent: Risks and Administration</u></p> <p>Administration of contrast agent should be considered in terms of risk-versus-benefit.</p> <p>Before the patient undergoes an examination using intravascular iodinated contrast media</p> <ol style="list-style-type: none"> a) Confirm that the intravascular administration of iodinated contrast media is critical to the clinical management of the patient. b) Consider whether an examination without contrast, or a different examination with lower risk, can be used as an alternative. <p>If administration of intravascular contrast media is required, and agreed by the radiologist responsible:</p> <ol style="list-style-type: none"> a) Use the smallest dose possible to answer the clinical question b) Consider using iso-osmolar contrast agents c) Avoid repeating intravascular contrast procedures d) Temporarily discontinue diuretics, ACE-Inhibitors and Angiotensin-II Receptor Blockers if there are no contra-indication (such as heart failure) for 24 hours before and after the procedure, if in doubt discuss with senior clinician e) Temporarily discontinue NSAIDs and metformin for 1-2 days before and after procedure f) Administer i.v. Sodium Chloride 0.9% (1ml/kg/hr) for 12 hours pre- and post-procedure unless contra-indicated (e.g. the patient is fluid overloaded or in cardiac failure). g) Further exposure to contrast media should be delayed until full recovery of renal function unless absolutely necessary. h) Renal function should be checked up to 48-72 hours following the procedure in a high risk group to ensure stable renal function.
<p>3.6</p>	<p><u>Assessment of the Patient with AKI</u></p> <p>History and Examination of the Patient with AKI</p> <ol style="list-style-type: none"> a) Try to identify whether the patient has pre-renal, intrinsic renal or obstructive AKI. <ul style="list-style-type: none"> • Antecedent illness or surgery • Systemic symptoms (fatigue, fever, weight loss) • Difficulty passing urine • Reduced urine output • Symptoms of infection b) Record the patient's drug history.

	<p>c) Specifically enquire whether the patient has taken NSAIDs, herbal or alternative medications (these are often bought 'over the counter' and not always volunteered with the drug history).</p> <p>d) Look for any previous biochemistry results to establish whether there is background chronic kidney disease (CKD).</p> <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>
3.7	<p><u>Determination of likely cause of AKI</u></p> <p>AKI in a hospital setting is often multifactorial. It is still useful to consider AKI in terms of:</p> <p>a) Pre-renal failure</p> <p>This is a decreased glomerular filtration rate resulting from renal hypoperfusion. 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 promote AKI.</p> <p>b) Intrinsic' Renal Failure</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 Failure</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. 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.</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 iLab pathology system.</p> <p>75% of AKI is due to either pre-renal failure or acute tubular necrosis (ATN)⁹ and quick diagnosis and treatment may prevent the need for dialysis and the associated increase in morbidity and mortality that results. Hospital acquired AKI is frequently multifactorial.</p>

	<p>e) Consider the following when investigating the potential causes of AKI</p> <ul style="list-style-type: none"> • Volume depletion • Sepsis • Medications which could be injurious to the kidney • Obstruction • Contrast Agent • Intrinsic Renal Disease • Rhabdomyolysis <p>UHL have adopted the use of an AKI Alert sticker (Appendix One) to promote early recognition of potential causes of AKI and encourage timely management using the AKI Care Bundle Actions (Appendix Two). (These can be ordered from the Print room ref no: AKI01)</p>
<p>3.8</p>	<p><u>Investigation of the causes of AKI</u></p> <p>In order to aid detection of the cause(s) of AKI all patients with AKI should have the following:</p> <p>a) Urinalysis</p> <ul style="list-style-type: none"> • Perform urine dipstick testing for blood, protein, leucocytes, nitrites and glucose in all patients as soon as acute kidney injury is suspected or detected. Document the results 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 has urine dipstick results showing haematuria and proteinuria, without urinary tract infection or trauma due to catheterisation. Send urine for MSU and urine albumin:creatinine ratio. <p>b) Blood Tests</p> <ul style="list-style-type: none"> • Biochemistry screen (U&Es, bone biochemistry, LFTs, bicarbonate, CRP, glucose) • FBC • Clotting screen • Blood cultures <p>c) Other Investigations</p> <ul style="list-style-type: none"> • ECG • CXR <p>d) Review of Nephrotoxic Medications</p> <p>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.</p> <p>e) USS renal tract</p> <p>Do not routinely offer an ultrasound of the urinary tract when a plausible cause of AKI has been identified.</p> <p>A renal ultrasound is useful to look for signs of obstruction and renal size (small scarred kidneys signify chronic kidney disease; asymmetric kidneys suggest renal artery stenosis or a non-functioning kidney).</p> <ul style="list-style-type: none"> • 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).

	<p>f) Where the history is suggestive, patients should also be tested for:</p> <ul style="list-style-type: none"> • 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 and Immunoglobulin in all patients who may have myeloma (elderly, anaemia or hypercalcaemia) • Urinary Bence-Jones protein (electrophoresis) for all patients who may have myeloma • Antistreptolysin 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 • Arterial blood gases for acid-base balance or gas exchange if any evidence of respiratory or cardiovascular compromise or low venous bicarbonate
<p>3.9</p>	<p><u>General Management of AKI</u></p> <p>The UHL AKI Care Bundle can be found in Appendix Two</p> <p>a) Monitoring patients with AKI</p> <p>The following should be recorded for all patients with AKI:</p> <ul style="list-style-type: none"> • 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 • Strict input and output recorded on fluid balance charts including losses from GI tract, drains etc. • Daily blood tests – U&E's, bone and venous bicarbonate • Daily weight • Ensure adequate nutrition <p>b) Treatment</p> <p>General treatment-refer to the AKI Care Bundle,(Appendix 2) priorities in treating AKI are:</p> <ul style="list-style-type: none"> • Early identification of underlying & remediable causes • Assess fluid status and optimise fluid balance (Intravenous fluids are the only reliable means of renal protection¹¹). 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 <p>Daily review by the medical team responsible for the patient is imperative.</p> <p>C) Assess Fluid Status</p> <p>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.</p> <ol style="list-style-type: none"> a) If the patient is obviously volume deplete then intravenous fluids should be initiated without the immediate need for CVP monitoring and fluid status 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 chronic kidney disease). If in doubt seek senior advice.

	<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) It is imperative that fluid status is regularly reassessed and that a strict record of input and output is maintained.</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) – 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, 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>
3.10	<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 still used to try and increase urine output and ease the degree of fluid overload and hyperkalaemia. However meta-analysis has shown that furosemide gave no benefit in terms of overall mortality, the need for RRT or the duration of RRT¹¹. Furthermore high doses can be associated with an increased risk of ototoxicity.</p> <p>Therefore whilst furosemide can be used for a short time for volume control in AKI, its use should not delay referral to Nephrology or the instigation of RRT when required and large doses should be given with caution.</p> <p>Furosemide and 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 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 Several randomised studies have shown that N-acetylcysteine is ineffective at preventing AKI.</p>
3.11	<p>Nephrology Advice</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 <p>The following patients should be discussed with the on-call Nephrology SpR (bleep through switchboard)</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>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 support¹⁰.</p>
3.13	<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>
3.14	<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>

3.15	<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>Patients should be referred for nephrology follow up if:</p> <ul style="list-style-type: none"> Renal function does not return to baseline according to UK CKD Guidelines¹¹ Patients have pre-existing CKD where referral should follow UK CKD Guidelines¹¹.
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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 AKI	Dr Richard Baines (Consultant Lead for AKI)	Case note audit	Quarterly for 2016 and annually thereafter	AKI T&F Group
Compliance with the AKI Care Bundle	Dr Richard Baines (Consultant Lead for AKI)	Case note audit	Quarterly for 2016 and annually thereafter	AKI T&F Group
Communication of AKI to Primary Care	Dr Richard Baines (Consultant Lead for AKI)	Audit of Discharge Letter	Quarterly for 2016 and annually thereafter	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

1. DOH (2005): Chronic Kidney Disease, Acute Renal Failure and End of Life Care <https://www.gov.uk/government/publications/national-service-framework-kidney-disease>
2. National Confidential Enquiry into patient Outcome & Death (2009) Acute Kidney Injury: Adding insult to Injury <http://www.ncepod.org.uk/2009aki.htm>
3. Duke GJ. Renal protective agents: a review. Critical Care & Resuscitation 1999; 1: 265-275
4. National Institute of Clinical Excellence (2013) Acute kidney Injury: prevention, detection, management. <https://www.nice.org.uk/Guidance/CG169>
5. KDIGO (2012) Clinical Practical Guideline for Acute Kidney Injury. Vol. 2, Issue 1. <http://www.kidney-international.org>
6. NHS England AKI Algorithm (2014) <https://www.england.nhs.uk/patientsafety/akiprogramme/aki-algorithm>
7. Powell-Tuck J, Gosling P, Lobo DN, et al (2008). British Consensus Guidelines on Intravenous Fluid Therapy for Adult Surgical Patients (GIFTASUP)
8. DOH (2000) Comprehensive Critical Care: A review of adult services.
9. Nash K, Hafeez A, Hou S. Hospital acquired renal insufficiency. Am J Kidney Dis. 2002; 39: 930-936)
10. National Institute of Clinical Excellence (2006) Nutrition support for adults: oral nutrition support, enteral tube feeding and parenteral nutrition (CG32) <https://www.nice.org.uk/Guidance/CG32>
11. Renal Association: Chronic kidney disease in adults: UK guidelines for identification, management and referral. London: Renal Association, 2005 www.renal.org/eGFR/refer.html

8. Key Words

Acute Kidney Injury, AKI, Staging, Care Bundle

DEVELOPMENT AND APPROVAL RECORD FOR THIS DOCUMENT	
Initial Author / Lead Officer: Dr Rachael Westacott Reviewed by: Dr Richard Baines Carole Best	Job Title: Consultant Nephrologist Consultant Nephrologist & AKI Lead AKI CQUIN Nurse
Approved by: Policy and Guideline Committee	Date Originally Approved: July 2009
Latest Approval Date: PGC 21 June 2019	Reviewed June 2019. Next review Date: June 2022
Version Number: 3	Details of Changes made during review: V1 Reviewed by the AKI Task & Finish group 4/2/16 <ul style="list-style-type: none"> • Changes made to format of the Guideline in keeping with new UHL policy & guidelines format. • New definition and staging system in place. • More emphasis on fluid assessment and reassessment & seeking senior advice regarding withholding rather than stopping nephrotoxic medication. • Management of AKI now grouped into AKI Care bundle • Information regarding discharge and follow up and information given to patients & carers included.

Appendix One – 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)