Welsh Acute Kidney Injury Group

All Wales Acute Kidney Injury (AKI) Guideline

A Quick Reference Guide for the Assessment and Management of AKI in Hospitalised Adult Patients in Wales

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With thanks to the Multidisciplinary Multispecialty Welsh AKI Group for supporting the development of this guideline, and the 2015 Steering Group for the previous All Wales AKI Guideline.

We welcome feedback and suggestions for improvement from colleagues across clinical areas. Should you wish to make any comments or join the AKI group, please email to Timothy.Scale@wales.nhs.uk and Sacha.Moore@wales.nhs.uk.

Rhwydwaith Arennau Cymru
Welsh Kidney Network

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GIG CYMRU NHS WALES

Table of Contents

Acute Kidney Injury (AKI) Quick Reference Bundle – think 'ROUNDUP'

R epeat U&Es

Include a bicarbonate or a venous blood gas.

O bstruction ruled out

Bladder scan and urinary tract imaging.

U rinalysis

Document a urine dip (blood/protein) in the medical notes.

N EWS2

Consider co-existing sepsis.

D ehydrated or overloaded?

Fluids if dry. If overloaded – senior medical/renal review.

U rine output

Ensure an accurate fluid balance chart with hourly urine output.

P rescriptions reviewed

May need to hold nephrosensitive medications or dose adjustment to medications cleared by kidneys.

Nephrology Referral Checklist for Acute Kidney Injury

To facilitate a concise yet thorough discussion and ensure the appropriate outcome is agreed upon, **please ensure you have the following information ready in front of you** prior to picking up the phone to nephrology and **consider the question you're asking**.

Demographics

- Patient's NHS number
- Patient's name & date of birth
- Hospital & ward you're calling from
- Your name, grade and contact number

Situation

- "I have a ...-year-old patient with a stage ... AKI (+/- hyperkalaemia/overload/acidaemia) on whom I would like advice regarding work-up/management/suitability for transfer/dialysis"

Background

- Past medical history (inc. underlying renal disease/transplant/risk factors for AKI)
- Medications (*tip - have the drug chart in front of you*)
- Functional baseline, frailty and pre-agreed ceilings of care

Assessment

- Concise clinical history, working diagnoses & treatments
- Baseline, admission and current creatinines *(tip - ensure you use creatinine rather than eGFR)*
- Examination findings, observations, volume status, urine output (*tip - look at the patient yourself prior to calling*)
- Up-to-date blood gas and electrolytes
- Urine dip result
- US KUB result

Recommendation

- Your differential diagnosis for the cause(s) of the AKI
- Any treatments you've already administered/actions you've taken for the AKI
- Any additional tests pending/you plan on sending
- **Your specific question(s) to the renal team**

Emergency Management of Hyperkalaemia with AKI in Hospitalised Adults in Wales

Ensure local guidelines are consulted.

Attached patient addressograph here.

Date: ___/___/___ Time: ___:___ Clinician:_____

Potassium (K+) (mmol/L):	Bicarb (mmol/L):	Urea (mmol/L):
pH:	pCO2 (kPa):	Creat (umol/L):

Full clinical assessment using an ABCDE approach + 12-lead ECG
VBG to confirm acid-base status
Consider pseudohyperkalaemia (utilise Lithium Heparin tube to exclude)

MILD
K^+ 5.5-5.9mmol/L
Review medications & diet
Manage AKI
Does not usually require specific treatment

MODERATE
K^+ 6.0-6.4mmol/L
Treat as per ECG, clinical condition & rate of K^+ change

SEVERE
K^+ ≥6.5mmol/L
Emergency treatment indicated

ECG Changes?

No

Yes

Protect the heart — 1

30mL 10% calcium gluconate *OR* ***calcium chloride** & repeat ECG

Temporary shift of potassium into cells — 2

IV Insulin-Dextrose infusion
CHECK BLOOD GLUCOSE FIRST
10 units soluble insulin (e.g. Actrapid) in 25g glucose (see glucose regimen list) over 15-30mins

If pre-treatment blood glucose <7.0mmol/L, the insulin-dextrose infusion should be **immediately** followed by **a further 25g glucose** (10% dextrose @50mL/hr for 5hrs)

*As an adjunctive therapy, **consider**:*

Nebulised Salbutamol 10-20mg
Reduce dose in tachyarrhythmia/heart disease

Remove potassium from the body — 3

Give Lokelma (Sodium Zirconium Cyclosilicate) 10g TDS PO for up to 72 hours (*or* Patiromer 8.4g OD PO) (in emergency drug cupboards). If overloaded consider IV diuretics

Monitor laboratory potassium & capillary (cap) glucose as per table, consider cause including medication review to prevent recurrence

If K^+ ≥6.5mmol/L despite medical therapy, liaise with nephrology/ICU regarding suitability for renal replacement therapy

(Adapted from the UK Kidney Association Guideline for the Emergency Management of Hyperkalaemia in Adults, 2020)

SUPPORTING INFORMATION

Complete <u>all three steps</u> rapidly

ECG Changes
Tented T Waves , Flat/absent p waves, Broad QRS , Sine wave pattern, Bradycardia, VT

IV Calcium (6.8mmol)
30mL 10% Calcium Gluconate IV over 10 minutes via large bore access
<u>OR</u>
***10mL 10% Calcium Chloride IV prefilled syringe or 7mL 1mmol/mL ampoule** over 5 minutes via large bore access

Repeat ECG, consider further dose after 5 minutes if changes persist.

Glucose Regimen (25g glucose)
=50mL 50% dextrose
=125mL 20% dextrose
=250mL 10% dextrose
Caution with large volumes in fluid overloaded patients

IV Sodium Bicarbonate
Outside of a critical care setting, and on the advice of a nephrologist/internal medicine physician/intensive care physician, 500mL 1.26% (or 1.4%) sodium bicarbonate can be administered IV over 2-4hrs if metabolic acidosis associated with hyperkalaemia.

Monitoring	Cap Glucose (mmol/L)	Potassium (mmol/L)
Baseline		
30min		
60min		
120min		
180min		
240min		
360min		

Definition

Acute Kidney Injury (AKI) is an abrupt reduction in kidney function usually diagnosed by an increase in the creatinine value. **AKI is not a diagnosis in its own right, rather a syndrome with many different underlying causes**. As such, it is important to diagnose and manage the underlying illness.

AKI is defined as:

- An increase in serum creatinine of ≥26.5 micromol/L within 48 hours; *OR*
- An increase in serum creatinine of ≥1.5x baseline creatinine which is known or presumed to have occurred within the last 7 days; *OR*
- A urine output of <0.5 mL/kg/hr for ≥6 consecutive hours

In most cases, AKI alerts on Welsh Clinical Portal (WCP) should prompt awareness of the presence of an AKI, however the creatinine trend should also be checked as the AKI alerts are not always accurate. Every AKI should be staged (see AKI Staging in Appendix 1) – this should be done automatically through WCP. Please note that **it is the creatinine which is used to diagnose and monitor AKI, not the eGFR**.

Any emergency hospital admission is at risk of AKI.

Clinical Assessment

A detailed history and A-E assessment are required in any unwell patient with AKI. Specific factors to consider in the history and examination are as follows:

History	Examination
Acute illness *e.g. infection*	Obs *e.g. blood pressure, heart rate, SpO2*
Volume losses *e.g. thirst, diarrhoea, burns*	Palpable bladder & bladder scan
Lower urinary tract symptoms	Rashes
Features of multisystem disease *e.g. arthralgia, nasal symptoms, haemoptysis, rashes, uveitis, weight loss, fatigue, fever, mouth ulcers*	Fluid status *e.g. mucous membranes, JVP, cap refill, skin turgor, thirst, peripheral oedema/ anasarca, weight, auscultation*
Medication history/toxin exposure *e.g. recently started medication, acute course, over-the-counter, recreational, long-term medications*	Evidence of pre-existing renal disease *e.g. fistula, dialysis catheter, transplant scar*
Risk factors in past medical history *e.g. CKD, diabetes, heart disease, cirrhosis*	Urine output
Family history of renal disease	Point-of-care ultrasound/echo if available

Diagnosis

Broadly, the pathophysiological mechanism of AKI can be divided into pre-renal, intra-renal and post-renal causes.

Pre-renal	Intra-renal	Post-renal
• Hypovolaemia • Hypotension • Hypoperfusion/ Renovascular	• Glomerular inflammation • Interstitial inflammation • Toxin-induced tubular damage	• Urinary tract obstruction

The most common cause is a pre-renal cause but, if this is sustained, the most common reason for a progressive or severe AKI is acute tubular necrosis (ATN). ATN will often result in an oliguric AKI that requires time and removal of the precipitant to allow for recovery (note a polyuric phase can occur during post-ATN recovery). AKIs are often multifactorial. Consider these potential causes when undertaking your clinical assessment and investigations.

Medication Review

There is no single approach to medications in AKI. A full medication review should be undertaken **in conjunction with a pharmacist** where possible, considering medications that may have contributed to the AKI alongside pre-existing medications which might need to be avoided/temporarily withheld/used with caution/therapeutic alternatives. The term 'nephrotoxic' is purposefully avoided and there is a **move towards the term 'nephrosensitive'**. The following are common medication culprits:

Consider avoiding	Consider temporarily withholding	Consider dose adjustment or switch to alternative
• Aminoglycosides *e.g. gentamicin* • NSAIDs *e.g. ibuprofen* • Trimethoprim (*n.b. may cause pseudo-rise in creatinine*) • Amphotericin	• ACEi *e.g. ramipril* • ARB *e.g. candesartan* • ARNI *e.g. Entresto* • Metformin • SGLT2 inhibitors *e.g. dapagliflozin* • MRA *e.g. spironolactone* • Diuretics *e.g. furosemide* ***if clinically dry***	• Anticoagulants *e.g. low molecular weight heparins, direct oral anticoagulants* • Opiates *e.g. switch morphine to oxycodone* • Gabapentinoids *e.g. gabapentin, pregabalin will accumulate* • Antimicrobials *e.g. dose reduce Tazocin, Aciclovir*

Investigations

The following investigations should be undertaken as standard in patients with AKI:

- **Blood tests** to include FBC, U&Es, LFTs, Bone Profile, CRP, Bicarbonate (if available).
- **Venous blood gas** (assessment of acid-base balance including lactate).
- **Urinalysis** (predominantly for blood & protein).
- **Blood cultures** and **urine MC&S** if clinical concern regarding infection/sepsis.
- **Bladder scan** (to exclude bladder outlet obstruction).
- **ECG** (for rhythm and in case of hyperkalaemia).
- **Chest x-ray** (for fluid overload/infection/pulmonary haemorrhage).
- **Urinary tract imaging** (US KUB or CT KUB if US not readily available) – perform within 6hrs if concern regarding obstruction/single kidney, or within 24hrs if high stage AKI/failure to response to initial treatment.

In those with a urine dipstick positive for blood and/or protein, or with unexplained or worsening stage 3 AKI, or with clinical concern regarding the possibility of intrinsic renal pathology (e.g. systemic symptoms), additional investigations may be warranted. **If advised to do so by a senior clinician**, some or all of the following investigations may be requested (frequently termed the 'renal screen'):

ANCA *(vasculitis)*	ANA, anti-dsDNA, C3, C4 *(SLE)*	Urinary ACR/PCR *(proteinuria quantification)*
LDH, blood film, reticulocytes (*TMA, if thrombocytopaenia)*	Anti-GBM *(Goodpastures)*	Serum electrophoresis & free light chains (urine Bence Jones if FLC not available), immunoglobulins *(myeloma)*
CK *(rhabdomyolysis)*	HbA1c *(diabetes mellitus)*	Virology *(Hep B/Hep C/HIV nephropathy)*

Additional investigations including ENA, cryoglobulins, ASOT, ACE, anti-PLA2R and urinary electrolytes may be requested under specialist guidance.

Management

The key to managing an AKI is to treat the underlying culprit. Key management principles to consider in all patients with AKI include:

- Accurate monitoring of input/output and daily weights.
  - Consider urinary catheterisation (particularly if concern regarding urinary retention).
- Review and adjust medications as per the Medication Review section.
- Rehydration with IV fluids *if volume deplete*.
  - The overall aim is to **target euvolaemia** in the majority of patients.
  - Repeated 250-500mL boluses of IV isotonic crystalloid fluid can be given as fluid challenges, initially up to 2L.
  - Crystalloid is also recommended for larger volume rehydration.
- Utilising diuretics *if clinically overloaded*.
  - Monitor response both in urine output and weight.
  - **For patients clinically overloaded with an AKI, an urgent review by a physician (SpR or above) should be undertaken.**
- Treat infection/sepsis promptly.
  - Appropriate antibiotics within one hour.
- Prompt management of hyperkalaemia.
  - This often coincides with AKI – see Hyperkalaemia Flowchart.
- Daily monitoring of urea, creatinine and electrolytes.

Additional management strategies will apply in special circumstances.

Indications for Nephrology Referral

The following are potential indications for referral to nephrology:

- Any stage 3 AKI, or unexplained or deteriorating AKI (regardless of stage)
- Patients with underlying CKD stage 4 or 5 (when appropriate)
- Possible glomerulonephritis (blood & protein on dip) or autoimmune disease
- Patients who are likely to need renal replacement therapy (RRT)
- All renal transplant patients
- All dialysis patients with hyperkalaemia or fluid overload

For advice on making a referral, see the Checklist for Nephrology Referral.

Renal Replacement Therapy (RRT) and Escalation of Care

If managed appropriately, the majority of AKI is reversible with medical management; however a small number of patients will require RRT.

The following are indications for renal replacement therapy:

- Refractory hyperkalaemia
- Refractory fluid overload/pulmonary oedema
- Refractory acidaemia
- Symptomatic uraemia (uraemic pericarditis or uraemic encephalopathy)
- Specific toxins in overdose (salicylate, lithium, ethylene glycol)

Medical management is likely to fail (even with initial improvements) if the patient is anuric.

Patients who are too unstable for transfer for renal input/renal replacement therapy may require RRT in a critical care setting. Consider referral to critical care for RRT in the following circumstances:

- All patients requiring CPAP/NIV/inotrope/vasopressor support.
- Multi-organ failure.
- GCS <12 or fluctuating.
- Patients unable to transfer from peripheral hospitals due to lack of bed capacity who require urgent, life-saving treatment, or unsafe to transfer.
- Systolic BP <90 mmHg (*discuss on case-by-case basis).
- FiO2 requirement >60% (*discuss on case-by-case basis).

Prior to initiating discussions with either nephrology or critical care about RRT, a decision should be made by a senior member of the parent team that escalation of care is appropriate and in the best interest of the patient.

Contrast-Associated Nephropathy (CAN) and Iodinated Contrast Use in AKI

In June 2023, the Royal College of Emergency Medicine and the Royal College of Radiologists issued a joint advisory statement on the use of intravenous iodinated contrast media in emergency computed tomography (CT) scanning. This recommended:

- **Patients requiring emergency iodinated intravenous contrast CT imaging should proceed to scanning without delay**. Specifically:
    - Measurement of renal function should not be considered a pre-requisite prior to scanning.
    - Pre-existing renal disease, diabetes mellitus or medication such as metformin should not delay scanning.
    - Age is not an independent risk factor for CAN and should not delay scanning.
    - Intravenous fluid administration should not be considered a pre-requisite.

In an emergency, a crucial diagnostic test or intervention requiring iodinated contrast media should not be delayed because of the renal function.

If intravenous iodinated contrast is required for a CT scan in an acute scenario, and if delaying the CT scan to allow time for potential optimisation of renal function is not feasible or may result in harm through lack of diagnosis and delay to targeted treatment, then the CT scan should go ahead with contrast regardless of the renal function. This does not routinely require discussion with nephrology.

The risk of developing CAN is potentially higher in those with an eGFR<60 ml/min (<30ml/min high risk), in those with diabetes mellitus, heart failure, volume depletion and cirrhosis. There is higher incidence with hyperosmolar iodinated contrast media (now rarely used in practice).

In all patients at high risk of CAN where imaging is required, consider:

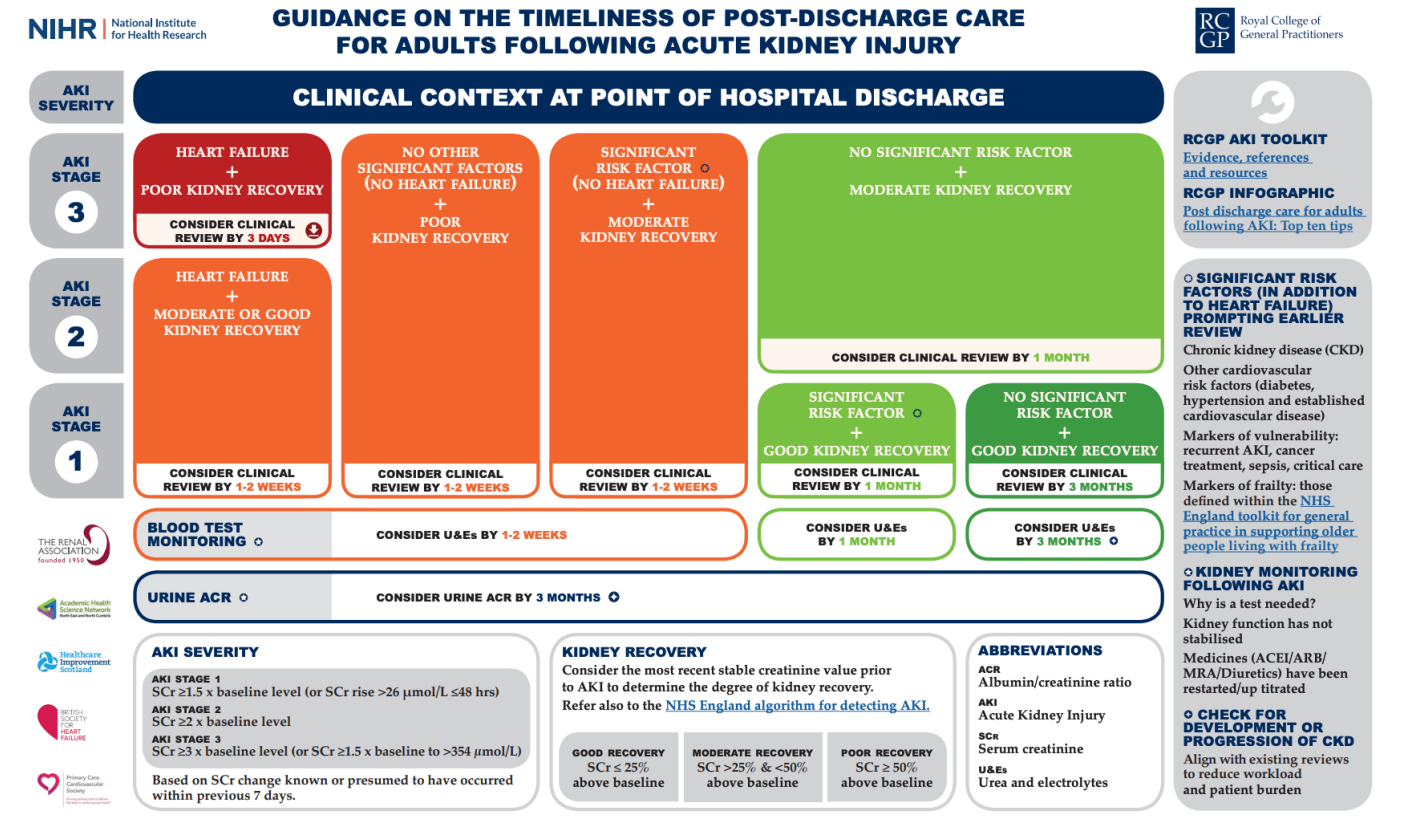
- Whether a contrast-enhanced scan will aid the acute management of the patient.
- Whether a non-contrast scan will answer the clinical question (consult a radiologist).
- Whether it is feasible and safe to delay imaging or treatment to facilitate optimisation of renal function prior to contrast utilisation.
- If contrast is necessary then, **in those at high risk of CAN, it is suggested that medications such as ACEi, ARBs and metformin are withheld for 24hrs pre- and post-contrast and hydration is optimised**, either with increased oral hydration or with 1L isotonic crystalloid over 12hrs pre- and post-procedure if clinically necessary.
- Patients at high risk for CAN should be informed about the potential risk by the requesting team and have follow-up U&Es checked 72hrs later.

Further information regarding the evidence base and controversies can be found here.

Discharge and Follow-Up

Following an episode of AKI, a plan should be made by the parent team (with specialist advice as necessary) for the re-review and, if appropriate, restarting of any medications which were temporarily withheld/adjusted due to the AKI.

The Royal College of General Practitioners has produced post-AKI follow-up guidance:



Communication is key. An accurate, succinct discharge summary is critical to ensuring primary care can undertake this follow-up.

As a minimum, the discharge summary should include: the peak AKI stage; the likely AKI aetiology(-ies); treatments administered; medications adjusted or withheld; specialist advice taken +/- RRT; the creatinine at discharge (compared with on admission).

NICE Quality Standard 76 advises a secondary care nephrology clinical review following acute kidney injury should be arranged where clinically/holistically appropriate:

- Within 90 days for those with residual CKD 4 at hospital discharge.
- Within 30 days for those with residual CKD 5 (non-dialysis) at hospital discharge.
- Within 30 days for those with ongoing dialysis requirements at the time of hospital discharge.

Patient Information and Sick Day Rules

The Kidney Care UK Patient Information leaflet on AKI can be provided to patients. Patients should be made aware that they have had an acute kidney injury with a brief overview of what this means and the relevant factors that contributed to this. Patients should also be made aware of any follow-up recommendations after discharge.

For patients taking potentially nephrosensitive medications, it is recommended that **sick day rules** counselling is considered and undertaken with the patient prior to discharge. Medications amenable to sick day rules discussions could include:

- ACE inhibitors (e.g. ramipril, perindopril) and ARBs (e.g. candesartan, irbesartan)
- ARNIs (e.g. Sacubitril/Valsartan (Entresto))
- SGLT2 inhibitors (e.g. dapagliflozin, empagliflozin)
- MRAs (e.g. spironolactone, eplerenone)
- Diuretics (e.g. furosemide, bumetanide, bendroflumethiazide)
- Metformin

Sick day rules counselling should be individualised. Patients may be advised to pause taking some of the above medications when they are acutely unwell and unable to eat and drink normally. This may be in the context of symptoms such as vomiting, diarrhoea, or fever.

Patients should be advised to avoid dehydration, monitor blood glucose if known to have diabetes mellitus, and contact a healthcare professional if their illness is not improving within 48 hours (or sooner if concerned).

Patients should be advised to restart these medications once they have recovered. As a suggested timeframe, this would typically be 24-48 hours after returning to normal eating and drinking.

Bibliography

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- The Renal Association Clinical Practice Guideline on Acute Kidney Injury (2019).

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Appendices

Appendix 1 – AKI Staging and NICE Standards for Response to AKI

If one of the above criteria is met, the AKI should be staged either by the degree of change in serum creatinine or the degree of change in urine output (whichever is more severe), as follows:

Stage	Serum Creatinine (SCr) Criteria	Urine Output Criteria
1	Increase ≥26 micromol/L within 48hrs *OR* increase ≥1.5 to 1.9x baseline SCr	<0.5 mL/kg/hr for ≥6hrs
2	Increase ≥2 to 2.9x baseline SCr	<0.5 mL/kg/hr for ≥12hrs
3	Increase ≥3x baseline SCr *OR* increase to ≥354 micromol/L *OR* commenced on renal replacement therapy (RRT) irrespective of stage	<0.3 mL/kg/hr for ≥24hrs *OR* Anuria for ≥12hrs

NICE Quality Standard 76 stipulates the following responses to AKI dependent upon stage:

- Adults with AKI stage 2 should have a clinical review within 6hrs if they are acutely ill or admitted to hospital, or within 24hrs if they are clinically stable.
- Adults with AKI stage 3 should have a clinical review within 6hrs or, if they are acutely ill in the community, an immediate review to consider hospital admission.

Appendix 2 – Supplementary Evidence around Iodinated Contrast Use in AKI

There is evidence that iodinated contrast media can rarely cause direct tubular toxicity. While there is some strength in evidence for intra-arterial contrast-induced nephropathy (e.g. with coronary angiography, perhaps due to first-pass renal exposure, although there are several confounders), there remains significant debate as to what extent contrast-associated AKI from small volume/iso-osmolar intravenous contrast administration (e.g. in CT scanning) is a clinically significant entity. It is accepted that in a small number of patients there may be a direct toxicity effect, and that IV contrast may contribute to a multifactorial AKI, however the large number of confounders and risk factors for AKI in patients undergoing imaging make studying this population problematic and the true figures are likely to be very small. **Recent large-scale observational data in patients with AKI has demonstrated that IV contrast administration was not associated with either persistent AKI at hospital discharge or initiation of dialysis within 180 days (Ehmenn et al, 2023).**

Appendix 3- AKI in Special Circumstances

The following are general tips for managing AKI in specific special circumstances. Further individualised advice should be sought from the appropriate specialty team.

Heart Failure

- Any form of heart failure can cause acute kidney injury.
- This is multimodal but often underpinned by venous congestion.
- These patients require specialist input (Nephrology/Cardiology/Internal Medicine) and often require **careful diuresis** and close monitoring.
- Diuretic resistance is an increasingly-recognised phenomenon in this cohort of patients. Again, those on high-dose loop diuretics with minimal decongestion should have specialist input from Nephrology/Cardiology.

Cirrhosis

- Creatinine is likely to be lower at baseline in patients with cirrhosis due to sarcopaenia, hepatic dysfunction and assay interference.
- **Not all AKIs in patients with cirrhosis are hepatorenal syndrome** (HRS). In fact, at least 60% are pre-renal and will respond to adequate volume resuscitation, medication optimisation and maintenance of euvolaemia.
- Common causes of AKI in cirrhosis include hypovolaemia (e.g. due to diuresis, laxatives), sepsis (e.g. spontaneous bacterial peritonitis), intra-abdominal hypertension from ascites, and GI bleeding.
- Potential cases of HRS (AKI with no response to diuretic withdrawal and fluid challenge with albumin and in the absence of: shock, 'nephrotoxic' medications, haematoproteinuria and normal urinary tract imaging) require early specialist input from Nephrology & Hepatology.

Immunotherapy

- Immune checkpoint inhibitors (ICPis) are increasingly used in the management of an array of malignancies and can cause immune-related adverse events (irAEs) in any area of the body. The incidence of a kidney irAE is ~1-4%.
- **A kidney irAE should be considered a differential in any patient on an immune checkpoint inhibitor**. Equally, other causes of AKI should still be considered.
- Early liaison with, and advice from, Oncology +/- Nephrology is required.
- Review guidelines for ICPi-associated AKI if available.

Rhabdomyolysis

- The breakdown of muscle resulting in myoglobinaemia (thus myoglobinuria and tubular heme burden) and volume depletion gives rise to AKI.
- Creatine kinase (CK) levels >5000 U/L identify patients at significant risk of AKI.
- The mainstay of treatment is to identify and treat the causative agent (usually drugs/trauma/seizures/long lie) and to keep the patient well hydrated with IV fluids (**typically giving enough fluid to target a urine output of ~200mL/hour**).
- Forced alkaline diuresis is not performed in routine practice.
- If CK levels do not fall, consider ongoing exposure to drugs, compartment syndrome or myositis as causes.

Myeloma

- Light chain cast nephropathy, light chain deposition disease and proximal tubulopathy are additional differentials to consider in those with AKI and known or suspected myeloma. Amyloidosis is another potential differential.
- It remains important to exclude other causes of AKI such as sepsis (particularly if on chemotherapy).
- **There is often good initial response to chemotherapy regimens that can improve renal function.** Adequate hydration with maintenance of a good urine output is also a mainstay of management in light chain cast nephropathy.
- Those with suspected renal involvement of their myeloma require early Haematology input.

Renal Transplant Patients

- AKI in renal transplant recipients can occur for the same reasons as in those without a transplant.
- Specific additional aetiologies to consider include: calcineurin toxicity, transplant obstruction, rejection, CMV/EBV/BK viropathy, increased risk of infection due to immunosuppression, vascular anomalies.
- **All transplant recipients with AKI should undergo an ultrasound of the transplanted kidney with doppler and have a trough level of immunosuppressive drug measured** (tacrolimus, ciclosporin).
- All cases of AKI in transplant recipients should be discussed with the renal/transplant team for individualised advice on work-up, treatment and immunosuppression.
- **Certain medications should not be given to patients on calcineurin inhibitors e.g. macrolides (clarithromycin), -azole antifungals.**