ACUTE KIDNEY INJURY (AKI) CLINICAL GUIDELINE:
SECONDARY CARE

TARGET AUDIENCE	Secondary care
PATIENT GROUP	All hospitalised patients

Clinical Guidelines Summary

- AKI is often multifactorial; 80% of AKI is due to pre-renal causes such as sepsis, hypovolaemia, hypotension and cardiac failure
- Key investigations for all patients with AKI:
  - FBC, U&E, LFT, CRP, bicarbonate, calcium, phosphate, lactate
  - Urinalysis +/- urine ACR
  - Renal ultrasound within 24h unless clear alternative cause or resolving
- Glomerulonephritis screen includes:
  - ANA, ANCA (MPO/PR3), rheumatoid factor, complement, anti-GBM
- Myeloma screen (if clinical suspicion) includes:
  - FBC, calcium, immunoglobulins & serum protein electrophoresis
  - Urine Bence-Jones protein
- Management of AKI:
  - Identify and treat the cause
  - Medication review – nephrotoxins, antihypertensives/diuretics, dose adjustment, therapeutic drug monitoring eg. digoxin, lithium
  - Fluid balance monitoring, catheter if oliguria/unable to reliably measure
  - Manage complications – see Hyperkalaemia guideline
  - Consider place of care – refer HDU/ITU if indicated
- Iodine-based contrast can be associated with AKI however AKI is not a contraindication – consider pre-hydration, discuss with Renal Team if concern
- When to refer to Renal via hospital switchboard:
  - Stage III AKI (Serum creatinine ≥3.0 x baseline)
  - Stage II AKI (2.0-2.9 x baseline) not responding to management
  - Proteinuria/haematuria, no clear cause for AKI, possible HUS/TTP, indication for urgent dialysis, AKI in renal transplant patient
- Indications for urgent dialysis:
  - Refractory: hyperkalaemia, metabolic acidosis, pulmonary oedema
  - Uraemic pericarditis/encephalopathy
- Reassess patient at point of transfer, if new hypotension/hypoxia/arrhythmia/ reduced consciousness, do not proceed with transfer & consider Critical Care

AKI Staging

Stage I
Creatinine 1.5–1.9 x baseline
Or creatinine increased by >26.5µmol/l
Or urine output <0.5ml/kg/hr for 6-12hrs

Stage II
2.0–2.9 x baseline
Or output <0.5ml/kg/hr for >12hrs

Stage III
≥3.0 x baseline
Or output <0.3ml/kg/hr for >24hrs
Or anuric for >12hrs

eGFR assumes creatinine is in steady state and is NOT valid in AKI.
AKI at any stage is associated with adverse outcomes. All AKI stage III and those worsening despite intervention (any stage) should be discussed with Renal.

Causes of AKI

Pre-renal

Any cause of hypoperfusion
- Sepsis
- Hypovolaemia
- Hypotension (incl. medications)
- Cardiac failure

80% of all AKI = pre-renal
Often multifactorial

Renal (intrinsic)

- Acute tubular necrosis
  - Prolonged pre-renal AKI
  - Rhabdomyolysis
  - Nephrotoxins
- Glomerulonephritis
- Vasculitis
- Myeloma
- Tubulointerstitial injury

Post-renal (obstructive)

- Prostatic enlargement
- Calculi
- Tumour (bladder, prostate, cervical)
- Retroperitoneal fibrosis

Clinical assessment

- History of fluid loss, recent intervention, medication changes
- NEWS – HR, BP, sats
- Urine output & fluid balance
- Weight (daily)
- Oedema, JVP
- Mucous membranes, skin turgor
- Chest auscultation (overload)
- Palpable bladder – retention
- Signs of infection
- Systemic features of vasculitis
- ↓Hb ↓platelets or haemolysis – consider HUS/TTP and discuss with Renal

Lead Author	Kaitlin Mayne, Zoe Cousland, Vivienne Li	**Date approved**	2022-05-16
Version	V1	**Review Date**	2025-05-16

Investigations

- **Urinalysis ± urine ACR if proteinuria**
- FBC, U&E, LFT, CRP, bicarbonate
- Calcium, phosphate
- Lactate
- Septic screen as guided by assessment
- CK if ?rhabdomyolysis/unexplained
- Consider glomerulonephritis (GN) and/or myeloma screen – see opposite
- HIV, Hep B, Hep C
- **Renal ultrasound within 24h unless clear alternative cause or AKI resolving** - if hydronephrosis discuss with Urology urgently

GN screen

if haematuria ± proteinuria

- ANA, ANCA (MPO/PR3)
- Rheumatoid factor, complement
- Anti-GBM

Myeloma screen

if clinical suspicion

- FBC, calcium
- Igs + serum protein electrophoresis
- Urine BJP/serum free light chains*

* SFLC not included in routine myeloma screen and must be specifically requested

Contrast-associated AKI

- Iodine-based contrast (CT/intervention) is potentially nephrotoxic however contrast-associated AKI is usually multifactorial
- **Renal impairment is NOT a contraindication to contrast** if benefit > risk
- If significant AKI or CKD 4-5, discuss with Renal team in case contrast may worsen AKI with potential to require dialysis
- Pre-hydrate with IV crystalloid if time allows and clinically appropriate
- Gadolinium-based contrast used for MRI is relatively contraindicated in AKI

Medication review in AKI

1. Stop **nephrotoxins** (gentamicin/NSAIDs)
2. Review **BP-lowering drugs**
3. Review all medications for **dose adjustment** & seek pharmacy advice eg:
    - Opiates
    - Anticoagulation
    - Antiepileptics
    - Antibiotics
4. **Monitoring of drug levels**: digoxin, theophylline, lithium, tacrolimus
5. **Any recent antibiotics?** Trimethoprim (& co-trimoxazole) can cause rise in creatinine (inhibits tubular secretion) without change in GFR ≠ not AKI and can also cause hyperkalaemia

Note: The Renal Drug Handbook/database is a useful resource for drug dosing.

Lead Author	Kaitlin Mayne, Zoe Cousland, Vivienne Li	**Date approved**	2022-05-16
Version	V1	**Review Date**	2025-05-16

Management

- **Find and treat the cause – AKI is not a diagnosis in itself**
- Relieve obstruction: catheter, Urology if nephrostomy/stenting may be indicated
- Correct hypovolaemia with IV crystalloid **fluids are not indicated if euvolaemic**
- If hypotensive, withhold antihypertensives, consider vasopressors/inotropes if replete
- Review medications considering clinical fluid status – **diuretics may be required if overloaded despite impaired renal function, may in fact need increased dose**
- Treat infection
- Monitor fluid balance – **catheter if oliguria/unable to reliably measure output**
- Manage complications
    - Hyperkalaemia (see guideline)
    - Acidosis (IV sodium bicarbonate as advised by Renal team/senior clinician)
    - Overload/pulmonary oedema – diuretics/GTN ± dialysis/ultrafiltration (Renal)
- Consider place of care – refer HDU/ITU if indicated

When to refer to Renal

1. Stage III AKI
2. Stage II AKI not responding
3. Proteinuria/haematuria
4. No clear precipitant for AKI
5. Possible HUS/TTP
6. Indication for urgent dialysis
7. AKI in transplant patient

When NOT to phone Renal first

- Obstruction → Urology
- Haemodynamic instability or multi-organ failure → HDU/ICU

Making the Renal referral

Contact Renal unit trainee via switchboard @ Monklands (consultant available 24/7)

Try to have the following available:

1. Bloods (see 'investigations')
2. Baseline renal function
3. Urinalysis
4. Fluid balance chart, weights
5. NEWS chart
6. Drug chart
7. PMH & pre-morbid function
8. TELP/escalation plan (own team)
9. USS report (if obtained)

Urgent dialysis if:

Refractory:

- Hyperkalaemia >6.5mmol/l
- Metabolic acidosis pH<7.15/H^+>70
- Pulmonary oedema
- Uraemic pericarditis/ encephalopathy

Also specific poisoning indications

Interhospital transfer

If there is a delay after being accepted by the Renal team, the **patient MUST be reassessed at point of transfer**.

If new hypotension, hypoxia, arrhythmia or ↓GCS DO NOT proceed – re-discuss ± consider Critical Care review

Lead Author	Kaitlin Mayne, Zoe Cousland, Vivienne Li	**Date approved**	2022-05-16
Version	V1	**Review Date**	2025-05-16

Appendices

1. Governance information for Guidance document

Lead Author(s):	Dr Kaitlin Mayne, Specialty Registrar Dr Zoe Cousland, Consultant Nephrologist & Clinical Lead Renal Medicine Dr Vivienne Li, Consultant Nephrologist
Endorsing Body:	Dr Mehrdad Malekian, Clinical Guidelines Team
Version Number:	V1
Approval date	2022.05.16
Review Date:	2025.05.16
Responsible Person (if different from lead author)	Dr Zoe Cousland, Consultant Nephrologist & Clinical Lead Renal Medicine

CONSULTATION AND DISTRIBUTION RECORD	
Contributing Author / Authors	Dr Kaitlin Mayne, Specialty Registrar Renal & General Internal Medicine; Dr Zoe Cousland, Consultant Nephrologist & Clinical Lead Renal Medicine; Dr Vivienne Li, Consultant Nephrologist.
Consultation Process / Stakeholders:	The guideline was developed in collaboration with NHS Lanarkshire Renal and Acute Medicine Physicians and has been approved by all members of the Renal Medicine Team.

Distribution	The guideline will be distributed via the NHS Lanarkshire Clinical Guidelines website and app.

CHANGE RECORD

Date	Lead Author	Change	Version No.
2022.05.16	Kaitlin Mayne, Zoe Cousland, Vivienne Li	Original guideline	1
			2
			3
		.	4
			5