

Causes of AKI

Pre-Renal AKI - decreased perfusion of the kidneys:

- **Volume depletion** (excessive diuresis, haemorrhage, dehydration, burns)
- **Cardiovascular disorders** (congestive cardiac failure, hypotension)
- **Obstruction of renal arteries** (renal thrombosis, renal artery stenosis)

Post-Renal AKI - obstruction to urine outflow, from the collecting ducts in the kidney down to the urethra.

- **Deposition of crystals** in the tubules, e.g. uric acid, sulphonamides, aciclovir, cisplatin.
- **Renal stones** in the ureter or bladder.
- **Tumour**, either within the tract or pressing on it from another pelvic organ, e.g. prostate hypertrophy, bladder cancer, bowel cancer.

Intra-Renal AKI - damage to the kidney itself

- **Sustained hypoperfusion**, or exposure to **nephrotoxic agents**. Antibiotics - aminoglycosides, amphotericin. Analgesics - salicylates. Ethylene glycol.
- **Autoimmune renal disease** - vasculitis, SLE, interstitial nephritis, glomerulonephritis, etc.

Response to AKI

ROUNDUP
In Acute Kidney Injury (AKI)
if the creatinine jumps by 26
do **ROUNDUP**

- R** Repeat U+E's
Daily U+E's whilst in AKI
- O** bstruction ruled out
Examine/bladder scan, US if no improvement
- U** rinalysis - dip urine
If blood/protein - send for urine PCR
- N** EWS Score 5
Consider co existing sepsis
- D** ry or wet ?
Consider rehydration, If fluid overload d/w renal/senior
- U** rinary output
Fluid balance chart
- P** rescriptions reviewed
Dose adjustments/consider if medications clinically appropriate

Escalation...
If K⁺ > 5.7 d/w renal/senior
If AKI 3 d/w renal/senior

High-Risk Medicines and Actions

When a patient is admitted with AKI, a thorough review of medication is required:

- To eliminate potential causes/contributory factors for AKI
- To avoid inappropriate combinations of medicines in the context of AKI
- To ensure all prescribed medicines and doses are clinically appropriate.

Review all Medications

- Remember to check medication history thoroughly and ask about "Over the Counter" preparations, herbal remedies or teas and alternative therapies.
- Check use of recreational drugs (cocaine, ketamine, etc).
- Consider withholding nephrotoxic medications on admission in patients at high risk of AKI.
- Ensure that all doses are tailored to the patient's degree of kidney impairment. Re-assess daily until AKI resolves.
- Educate the patient before discharge; which medications to restart and when.
- Discuss appropriate medicines to avoid in future and "sick day" guidance.
- Ensure information on which medications to restart and when are communicated to the GP or next care setting.

KDIGO Staging System for Acute Kidney Injury		
Stage	Serum creatinine	Urine output
1	rise ≥ 26 $\mu\text{mol/L}$ within 48hrs or rise ≥ 1.5 - to 1.9 X baseline SCr	<0.5 mL/kg/hr for > 6 consecutive hrs
2	rise ≥ 2 to 2.9 X baseline SCr	<0.5 mL/kg/hr for > 12 hrs
3	rise ≥ 3 X baseline SCr or rise 354 $\mu\text{mol/L}$ or commenced on renal replacement therapy (RRT) irrespective of stage	<0.3 mL/kg/hr for > 24 hrs or anuria for 12 hrs

Acute Kidney Injury (AKI) Medicines Optimisation



AKI is a rapid deterioration in a patient's kidney function over hours or days secondary to an acute event. It is a marker of deterioration in a patient and their reduced ability to compensate to a trigger factor.

- 65% of AKI starts in community
- In the hospital setting 20% of acute admissions will develop AKI.
- Up to 30% of all cases of AKI are thought to be due to drugs.
- 5% of inpatients develop drug-induced renal impairment.
- AKI: risk and onset are associated with poor outcomes, and the implication is context-specific. Risk increases with risk factors e.g. heart failure, diabetes. Outcomes are poorer in the vulnerable patient: prolonged hospital stay, mortality, CKD.

www.ukkidney.org/rpg

	Effects on renal/fluid/electrolyte physiology	Change in the side effect profile when renal function is reduced	Action in presence of AKI
NSAIDs / COX II inhibitors	Altered haemodynamics within the kidney leading to underperfusion and reduced glomerular filtration		Avoid these agents in people at high risk of AKI
Opioid analgesics		Accumulation of active metabolites in AKI (especially morphine, pethidine and codeine) – increased incidence of CNS side effects & respiratory depression	Avoid long-acting preparations. Reduce dose and frequency. Use opiates with minimal renal excretion e.g. fentanyl, oxycodone, hydromorphone, tramadol
Pregabalin & Gabapentin		Accumulation leading to an increase in CNS side effects	Reduce dose
Antihypertensives	Hypotension may exacerbate renal hypo-perfusion	Risk of bradycardia with Beta Blockers	Consider withholding / reduce dose depending on blood pressure
ACEI / ARBs / Aliskiren	Hypotension / Hyperkalaemia		NOT nephrotoxic – can cause more harm than good if stopping. In AKI consider withholding / reduce dose if hyperkalaemia or hypotension
SGLT2I	Hypotension / volume depletion		Hold if AKI – remember sickday guidance
Diuretics (Thiazide & Loop)	Volume depletion. Acute interstitial nephritis (rare)	Loop diuretics preferred as thiazides less effective if GFR < 25ml/min. However thiazides can potentiate the effects of loop diuretics	If volume depleted, consider withholding
Potassium sparing diuretics amiloride	Volume depletion		Stop if AKI and hyperkalaemia
Eplerenone, spironolactone, finerenone	Hyperkalaemia		
Statins	May cause AKI if rhabdomyolysis is present	Increased risk of rhabdomyolysis	Stop if AKI due to rhabdomyolysis, OR if patient develops unexplained/persistent muscle pain
Digoxin	Hyperkalaemia	May accumulate in AKI leading to bradycardia, visual disturbances, mental confusion	Reduce dose. Monitor potassium and drug levels
Direct Oral Anticoagulants		May accumulate leading to increased risk of bleeding	Consider withholding, particularly agents with high renal clearance.
Aciclovir / Valaciclovir	Crystal nephropathy Acute interstitial nephritis (rare)	Drug accumulates in reduced renal function leading to mental confusion, seizures	Reduce dose. Encourage patient to drink plenty
Aminoglycosides	Tubular cell toxicity	Ototoxicity	Avoid if possible. If use is unavoidable, reduce dose &/or increase dosing interval. Monitor drug levels and renal function 2 – 3 times per week
Carbapenems		Drug accumulates in reduced renal function leading to mental confusion, seizures	Reduce dosing frequency
Fluconazole		Accumulation leading to acute mental confusion, coma, seizures	Reduce dose
Ganciclovir / Valganciclovir	Crystal nephropathy	Accumulation leading to neutropenia, anaemia and thrombocytopenia	Reduce dose. Monitor renal function and full blood count
Vancomycin	Acute interstitial nephritis (rare)	Accumulation leading to renal toxicity, ototoxicity	Reduce dose/increase dose interval. Monitor levels and renal function 2-3 times per week
Trimethoprim, Co-trimoxazole	Increased risk of hyperkalaemia (especially in combination with spironolactone or ACEI/ARB)	Accumulation increases risk of hyperkalaemia (particularly with high doses), nausea and vomiting	Avoid or reduce dose (particularly if patient is already taking an ACEI, ARB or spironolactone)
Phenytoin	Acute interstitial nephritis (rare)	Risk of phenytoin toxicity if patient has low serum albumin levels	Monitor levels. Correct phenytoin levels for uraemia and low serum albumin
Hypoglycaemic Drugs e.g. gliclazide		Accumulation in AKI may increase risk of hypoglycaemia	Avoid long-acting preparations. Monitor blood glucose levels & reduce dose if necessary
Metformin		Risk of lactic acidosis increased. Accumulation leading to hypoglycaemia	Avoid if GFR < 30 ml/min
Colchicine		Diarrhoea / vomiting	Use lower dose. AVOID NSAIDs
Lithium	Can cause nephrogenic diabetes insipidus	Accumulation increases risk of side effects	Avoid where possible. Monitor levels