

Practical guide to the definition and management of acute hyperkalaemia in individuals with cardiorenal disease on RAASi* therapy

Hyperkalaemia should be considered a predictable, recurrent and manageable issue in individuals living with cardiorenal disease and treated with RAASi therapy¹

ASSESSMENT**

	Mild hyperkalaemia	Moderate hyperkalaemia	Severe hyperkalaemia
Potassium level (mmol/L)²	5.5–5.9	6.0–6.4 (address as severe hyperkalaemia if >6.0 with electrocardiogram (ECG) changes)	>6.5
Exclude pseudohyperkalaemia² [BOX 1] (repeat sample in lithium heparin tube)	Within 3 days if unexpected (otherwise 14 days)	Same day	Urgent hospital assessment
Is patient acutely unwell? (include consideration of AKI [BOX 2])	Yes – urgent hospital assessment No – continue	Yes – urgent hospital assessment No – continue	Urgent hospital assessment
ECG²	No ECG required (unless clinical concern)	Urgent 12 lead ECG – ECG changes [BOX 3] – urgent hospital assessment No ECG changes – continue	Urgent hospital assessment
Where to assess and manage patient	Community likely appropriate	Same day emergency care via hospital	Urgent hospital assessment

BOX 1

Pseudohyperkalaemia²

Causes

Difficult venepuncture, haemolysis, thrombocytosis, erythrocytosis, prolonged or cold storage

Reducing risk

Label collection time, minimise transit time, optimise storage conditions

BOX 2

Acute Kidney Injury (AKI)

Causes³

- **Prerenal** – reduced blood flow to the kidney, e.g. haemorrhage, diarrhoea and vomiting, cardiogenic shock, nonsteroidal anti-inflammatory drugs (NSAIDs)
- **Renal** – conditions that affect the glomerulus or tubule, e.g. acute tubular necrosis, acute interstitial nephritis
- **Postrenal** – obstructive causes, e.g. renal/ureteric calculi, tumours, enlarged prostate

Diagnosis⁴

AKI is defined as any of the following:

- 1 Increase in serum creatinine (sCr) $\geq 26.5 \mu\text{mol/L}$ within 48 hours; or
- 2 Increase in sCr ≥ 1.5 times baseline, which is known or presumed to have occurred within the prior 7 days; or
- 3 Urine volume $<0.5 \text{ mL/kg/h}$ for 6 hours

BOX 3

ECG changes associated with hyperkalaemia²

Progressive changes:

- Tented T waves
- Prolonged PR interval, flat P waves
- Wide QRS, sine wave, arrhythmia, cardiac arrest

*Renin Angiotensin Aldosterone System inhibitor therapy (ACE inhibitors, angiotensin II receptor blockers, angiotensin receptor/neprilysin inhibitor, mineralocorticoid receptor antagonists)

**Decisions to admit and the route of admission should be based on clinical judgment and local resource, with due consideration of any relevant variables outside of the scope of this guide

MANAGEMENT**

Management approach

If acutely unwell – urgent hospital assessment

If well – see '**Potassium lowering strategies and therapies**'

Continue RAASi therapy if possible – see '**Potassium lowering strategies and therapies**' and '**RAASi therapy in the context of hyperkalaemia**'

If acutely unwell or ECG changes – urgent hospital assessment

If well – see '**Potassium lowering strategies and therapies**'

Continue RAASi therapy if possible – see '**Potassium lowering strategies and therapies**' and '**RAASi therapy in the context of hyperkalaemia**'

Follow local hospital protocol e.g. UK Kidney Association (UKKA) 5-step approach²

1. **Protect the heart** (calcium gluconate, calcium chloride)
2. **Shift potassium into cells** (insulin dextrose, salbutamol)
3. **Remove potassium from the body** (potassium binder)
4. **Monitor potassium and glucose**
5. **Prevent recurrence** – see '**Potassium lowering strategies and therapies**'

Withhold RAASi therapy. Once normokalaemia achieved, re-initiate and re-optimize RAASi therapy if possible. See '**Potassium lowering strategies and therapies**' and '**RAASi therapy in the context of hyperkalaemia**'

Approach to RAASi therapy

Potassium lowering strategies and therapies:

1. Address correctable causes of hyperkalaemia:

- ✓ **Adjust potassium elevating drugs**^{1,2} – Prioritise those that can be swapped/withheld with least adverse consequences. **Down-titrate/discontinue RAASi therapy as a last resort**⁵ – see '**RAASi therapy in the context of hyperkalaemia**'
- ✓ **Modify diet** – Advise a healthy, diverse diet with higher consumption of plant-based foods than animal based foods and low consumption of ultra processed foods⁵. If potassium remains >5.5 mmol/L once non-dietary factors are addressed, refer to specialist dietician²
- ✓ **Correct metabolic acidosis**² – Metabolic acidosis increases the risk of hyperkalaemia
- ✓ **Optimise glycaemic control**² – Poorly controlled diabetes increases the risk of hyperkalaemia
- ✓ **Avoid/address constipation**² – Constipation increases the risk of hyperkalaemia

2. Consider potassium lowering medications (acutely or longer term):

- ✓ **Diuretics** – Can increase potassium excretion
- ✓ **Bicarbonate** – Consider adding oral sodium bicarbonate if serum bicarbonate <22 mmol/L
- ✓ **Potassium binders** – Remove potassium from the body via the gastrointestinal tract²

3. Prevent recurrence of hyperkalaemia:

- ✓ Recurrence of hyperkalaemia should be anticipated, and steps taken to avoid it²
- ✓ Careful prescribing of potassium elevating drugs – use only where clearly indicated, with particular care if combinations are required, e.g. ACEi/ARB/ARNi + MRA for heart failure (HF)²
- ✓ Regular review of correctable causes and consideration of the need for potassium lowering medications (as above)²
- ✓ Regular monitoring of bloods (potassium and renal function) and review should occur at the frequency appropriate for the disease state and the individual, e.g. 1–4 times per year for chronic kidney disease (CKD)⁵ and HF⁶, with additional monitoring during intercurrent illness (especially dehydrating illness), titration of medications that affect potassium levels or renal function, or change in the underlying cardiorenal condition
- ✓ Education of individuals with cardiorenal disease

**Decisions to admit and the route of admission should be based on clinical judgment and local resource, with due consideration of any relevant variables outside of the scope of this guide

✗ **Sick day guidance** – A temporary pause of RAASi therapy, diuretics, metformin and sodium–glucose co–transporter–2 inhibitors during acute dehydrating illness may decrease the risk of AKI and hyperkalaemia. However, the evidence base for this is weak and there is potential for harm if these medications are not re–instated. **Sick day guidance should be based on an individual risk assessment and there must be a clear plan to re–instate any paused medications.**^{2,5}

†Potassium elevating drugs²

- RAASi (ACE inhibitors, angiotensin II receptor blockers, mineralocorticoid receptor antagonists)
- Potassium supplements
- Potassium–sparing diuretics
- Trimethoprim/co–trimoxazole
- NSAIDs
- Non–selective beta–blockers
- Antifungals
- Digoxin
- Salt substitutes
- Herbal medicines (e.g. alfalfa, dandelion)

RAASi therapy in the context of hyperkalaemia:

- Hyperkalaemia associated with RAASi use can often be managed by measures to reduce potassium other than down–titration or discontinuation of RAASi therapy⁵
- Down–titration or discontinuation of RAASi therapies is associated with adverse clinical outcomes in CKD and HF^{7–10}
- Only down–titrate or discontinue RAASi as a last resort; if hyperkalaemia is uncontrolled despite **'Potassium lowering strategies and therapies'**, or symptomatic hypotension or serum potassium >6.5 mmol/L (until normokalaemia achieved)⁵
- Re–initiate and re–optimise RAASi therapies that are down–titrated or discontinued once normokalaemia is achieved wherever possible – utilise appropriate **'Potassium lowering therapies and strategies'**
- If hyperkalaemia is preventing RAASi optimisation, seek specialist advice
- N.B. If RAASi therapies are discontinued, also discontinue potassium lowering therapies as appropriate

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