



## Inpatient Hyperkalaemia Guideline

After potassium enters the body, most is stored in cells, and then excreted in urine. Hyperkalaemia is usually due to **excessive intake** (particularly in patients with a degree of existing renal impairment), **increased release from cells**, or **reduced urinary excretion**.

Consider whether result is accurate. Potassium can be released from cells after sample taken e.g. haemolysed sample – consider a heparinised sample, particularly in patients with thrombocytosis

Consider why your patient may be hyperkalaemic

- Drugs: Commonly ACE inhibitors or angiotensin receptor blockers, spironolactone, trimethoprim, betablockers, calcineurin inhibitors, potassium supplements.
- Metabolic acidosis: hydrogen ion buffering promotes potassium movement to extracellular fluid to maintain electron neutrality
- Insulin deficiency/hyperglycaemia/hyperosmolality: insulin promotes potassium movement into cells. Hyperosmolality reduces intracellular water and the higher potassium gradient moves potassium out of cells. If the patient has DKA, refer to the DKA protocol.
- Tissues breakdown: trauma, rhabdomyolysis, tumour lysis syndrome, red cell haemolysis, exercise
- Hypovolaemia or reduced effective blood volume e.g. heart failure, liver failure
- Hypoaldosteronism: often coexistent hyponatraemia
- Acute kidney injury: consider referring to the AKI guidelines on Firstport

The urgency of treatment depends on degree of hyperkalaemia and signs/symptoms. Patients with hyperkalaemia due to tissue breakdown e.g. rhabdomyolysis need more aggressive treatment as they will continue to release potassium from damaged tissues despite treatment.

All patients with potassium >6 should have an ECG, although ECG changes do not necessarily correlate with the potassium level. ECG abnormalities associated with hyperkalaemia include tall T waves, short QT, long PR, broad QRS, bradycardia, VT, VF and asystole. **Patients with ECG changes require urgent treatment.**

Management:

1. Antagonise the effects of hyperkalaemia with calcium
  - **10ml of 10% calcium gluconate over 10 minutes** intravenously (ideally with cardiac monitoring if this can be arranged quickly – do not delay treatment if cardiac monitoring is not available in your ward).
2. Drive potassium into cells
  - **8 units actrapid** in 100ml of 20% dextrose given intravenously over 30 minutes
  - Nebulised **salbutamol** 5mg
  - Recheck glucose and potassium after 1 hour
  - In patients with acidosis and renal impairment, sodium bicarbonate may be of benefit but should be discussed with a senior medical or renal physician
3. Remove potassium from body
  - Improve urine output with fluids if hypovolaemic or loop diuretics if fluid overloaded

- **Calcium resonium** given orally or rectally promote excretion from the bowel
- Dialysis or haemofiltration in oliguric or anuric patients.
- 4. Ongoing monitoring
  - Insulin/Glucose infusion will only reduce the potassium transiently
  - Serum potassium should be rechecked 4-6 hours after infusion and at least daily until safely controlled and underlying causes addressed

When to discuss with renal team (ward 1, Monklands):

- Any chronic dialysis patient with hyperkalaemia, as medical treatment will not be effective and they need urgent dialysis
- Oligoanuric patients should be discussed as they may need acute renal replacement therapy
- Patients with rhabdomyolysis or tumour lysis as they may continue to release potassium despite medical treatment
- Patients in whom medical management has been ineffective after repeated insulin/dextrose infusions
- For patients outwith Monklands hospital who have refractory hyperkalaemia with ECG changes, it may be more appropriate to discuss with local ITU team for haemofiltration as interhospital transfer may add unsafe delays to treatment (please refer to the [AKI bundle](#) for further guidance on the transfer of patients between hospitals for renal replacement therapy).