Hyperkalaemia in dialysis and CKD patients

Hyperkalaemia can be a medical emergency but the risk of complications, including arrhythmias, is variable and difficult to define. The protocol presented below is a guide only; treatment must be individualised.

Manifestations of hyperkalaemia

- Sudden death
- Cardiac conduction abnormalities and arrhythmias
- Skeletal muscle weakness or paralysis

ECG changes

- Tall peaked T waves
- Widening of the QRS complex
- Loss of P wave leading to “sine wave” pattern
- Ventricular fibrillation or sinus arrest with asystole can result

Cardiac arrhythmias are the most critical complications. Factors that may increase the risk of cardiac arrhythmias include:

- Degree and rate of rise of the hyperkalaemia
- Pre-existing cardiac disease and ECG changes
- Other electrolyte abnormalities (eg. hypocalcaemia, acidosis)

Management of hyperkalaemia

Initial assessment after hyperkalaemia detected

- Repeat K (urgent).
  - Do not wait for result before starting treatment; this is to confirm that the result is not spurious and to give information about the rate of rise of the serum K
- Urgent ECG looking for manifestations of hyperkalaemia as above.
  - Note the degree of hyperkalaemia does not always correlate with ECG changes.
- All patients with ECG changes must be given i.v. Calcium to protect the myocardium and have continuous ECG monitoring to detect arrhythmias that might develop.

Potassium > 7.0 mmol/L and/or ECG changes

- Continuous ECG monitoring
- Intravenous Calcium gluconate (or chloride)
  - Cardioprotective; has no effect on [K]
  - In patients on digoxin must be given over 20 minutes as rapid infusion may precipitate myocardial digoxin toxicity.
  - Ca gluconate
    - 10 mls of 10% over 2-3 minutes.
    - Equivalent to 2.2 mmol of calcium
    - Effect immediate and lasts for 30 – 60 minutes
    - Repeat in 5 minutes if ECG changes persist
  - Ca chloride
- Dose 5-10 mls of 10% solution
- Equivalent to 3.4 to 6.8 mmol of calcium
- 3 times more calcium is available than calcium gluconate but is toxic to veins and should only be given via a central vein

- **Shift K into cells**
  - Insulin and glucose (Stimulates Na K ATPase)
    - 50mls of 50% glucose plus 10 Units of actrapid ivi
    - Onset 15-30 minutes and lasts 4-6 hours
    - Effect can be maintained by continuing an infusion of 10% glucose
    - Monitor BSLs every hour
    - Lowers K by 0.5-1.5 mmol/L
  - Salbutamol (Ventolin) (Stimulates Na K ATPase)
    - 5mg via nebuliser over 10-20 minutes
    - Onset 20-30 minutes and lasts about 2 hours.
    - Lowers K by 0.5-1.0 mmol/L
    - May not work as well in patients on dialysis and should not be used as sole measure in these patients.
    - Has an additive effect with insulin and glucose.
  - Sodium bicarbonate
    - Not generally used these days, except if patient very acidic. In these cases dialysis may be preferable
    - Some risks associated with its use, particularly fluid overload or tetany in patients with hypocalcaemia.
    - Works best if patient is acidic. (Some studies actually show no reduction in potassium)
    - Onset slower.
    - Dose 50mls of 8.4% NaHCO3 (50 mmols)
    - Be careful if patient overloaded
    - Must not be given in same iv as calcium.

- **Remove K from body**
  - This can be done through ion exchange resins or dialysis
  - Sodium polystyrene sulfonate (Resonium A) exchanges Na for K in the gut.
    - Mix15-30gm in water and give orally (or 30 – 60 gm PR if vomiting but not as effective)
    - Onset 1-2 hours
    - Continue q6h if necessary based on serum K
    - Cease once K under 5 as K may continue to fall for 1-2 days.
  - Calcium polystyrene sulfonate is also available but not generally used except in children. It exchanges calcium for K and can lead to hypercalcaemia

- **Contact the renal registrar on call (or nephrologist on call if registrar not contactable):**
  - This is important so that decision about the need for dialysis can be made early, particularly for chronic dialysis patients.
  - The need for dialysis needs to be individualised

- **Monitor closely**
  - Repeat the K and check the BSL at least every 1-2 hours till patient out of danger and K stable.
  - Continuous ECG monitoring whilst K being treated.

- **Investigate the cause**
  - Drugs
Addison’s Disease
- Volume depletion
- Acute deterioration in renal function etc

- Dietitian review for low K diet.

**K 6.5 – 6.9**
- Check ECG - Cardioprotection generally not necessary unless ECG abnormalities
- Shift K into cells
  - Insulin and glucose as above
  - Ventolin via nebuliser as above
- Resonium A
- Review possible causes

**K < 6.5 mmol/L**
- Not generally live threatening, particularly in patients with CKD
- Review diet and medications
- Check status of Renin and Aldosterone, particularly if renal disease not severe.
- For non-dialysis patients, renal excretion of K can be enhanced by:
  - Correcting volume depletion if present and encouraging patients to drink more.
  - Lasix 40-80 mg/day
  - Florinef (fludrocortisones) 0.1 mg/day
    - Watch for oedema
    - Avoid if heart failure
- If [HCO₃] under 22 mmol/L give oral NaHCO₃ (Sodibic) to correct.
- Needs review by dietitian