Acute Hyperkalaemia (High K)

Background

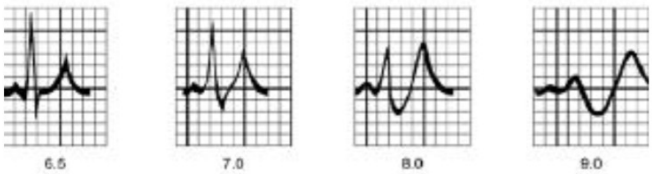
- Defined as of more than 6.0 mmol/L
- This is a 'Time Critical Emergency'
- Always think of "*pseudohyperkalaemia*" (haemolysis)
- Potassium secretion is proportional to flow rate and sodium delivery through distal nephron (diuretics will cause Low K
- Acidosis is associated with worsening
- Most medical treatment in the ED is temporising
- Urgent Dialysis may be required

Causes Differentials

- Pseudohyperkalaemia
- Crush Syndrome (trauma), release from cells (transfusion)
- Excess Intake
- Decrease Excretion
- Medications (see below)

First Test – Monitor patient and obtain ECG

- The classic ECG changes are not always present in true High K
- Progressive changes include:
  - *Flat/long P waves*
  - *Broad bizarre QRS complex*
  - *Slurring into the ST segment*
  - *Tall tented T waves*
  - *Long PR interval*



- In severe cases - sine waves, ventricular fibrillation, progressive heart block, asystole

Management Priorities

RESUSCITATION

- Call for assistance and assemble an appropriate team (nursing and medical). Move the patient to the Resuscitation Bay.
- Attach the patient to 3 lead ECG telemetry and observe with continuous non-invasive monitoring
- Apply O2 / salbutamol - Position Patient – Sit patient up
- Obtain IV access

- Send bloods (including a bedside point of care VBG)
- Start Intravenous (IV) fluid (e.g. 0.9% saline 500ml/h if not known 'renal patient' or a significant risk of fluid overload with small amounts of IV fluid) – this is safe and appropriate in most patients

SPECIFIC TREATMENT

(1) Immediate 12 lead ECG

(2) Stabilize cardiac membrane using Calcium

(3) Shift K+ into cells

a. IV insulin 10 units
b. 25-50 grams glucose (e.g. 50mls of 50% dextrose)
c. Check the blood sugar and assess the patient for neurological symptoms within 20 minutes
d. Nebulise Salbutamol (e.g. 10mg nebuliser)
e. If acidotic/critical – consider 8.4% sodium bicarbonate
    i. 50 ml of 8.4% solution over 10 minutes

(4) Remove K+ from system

a. In patients with adequate renal function - IV Frusemide 1mg/kg (40-80 mg)
b. Calcium Resonium / K Binding Resin – possibly very ineffective and **risk of complications** (see below)
c. IV normal saline solution for volume expansion if dry, crush syndrome or DKA
d. Definitive treatment for High K is haemodialysis

(5) Monitoring - do follow up ECGs and continuous telemetry

(6) Document the patient needs 'Low K+ Diet' & 'Fluid Balance Plan'

(7) Admission under renal team / ICU

Summary of Treatment Options

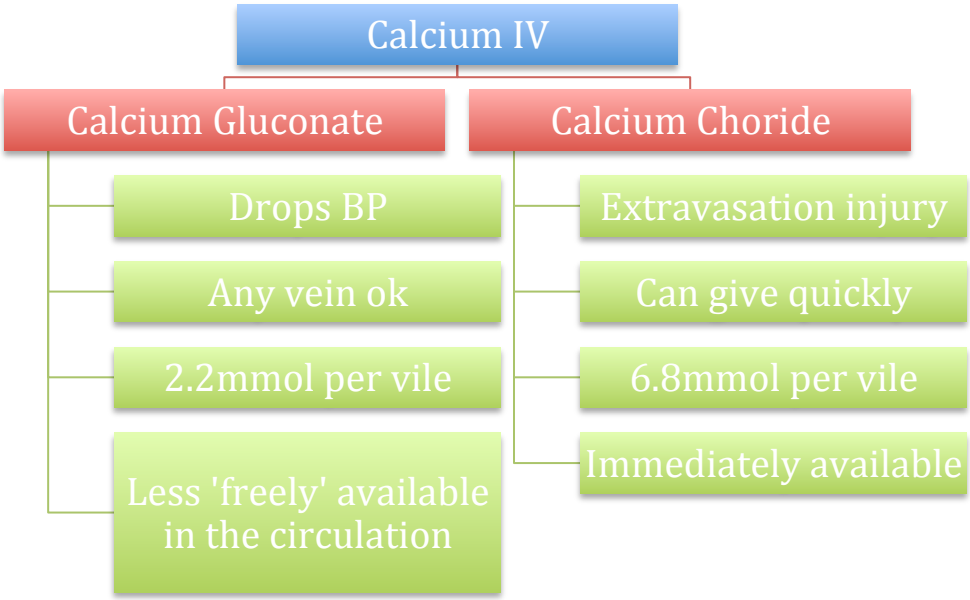
(Weisberg et al. Management of Severe Hyperkalemia. Crit Care Med. 2008 Dec;36(12):3246-51.)

Table 1. Emergency treatment of hyperkalemia

Agent	Dose	Onset	Duration	Complications
Membrane stabilization				
Calcium gluconate (10%)	10 mL IV over 10 min	Immediate	30–60 min	Hypercalcemia
Hypertonic (3%) sodium chloride	50 mL IV push	Immediate	Unknown	Volume overload hypertonicity
Redistribution				
Insulin (short acting)	10 units IV push, with 25–40 g dextrose (50% solution)	20 min	4–6 hrs	hypoglycemia
Albuterol	20 mg in 4 mL normal saline solution, nebulized over 10 min	30 min	2 hrs	Tachycardia inconsistent response
Elimination				
Loop diuretics				
Furosemide	40–80 mg IV	15 min	2–3 hrs	Volume depletion
Bumetanide	2–4 mg IV			
Sodium bicarbonate	150 mmol/L IV at variable rate	Hours	Duration of infusion	Metabolic alkalosis volume overload
Sodium polystyrene sulfonate (Kayexalate, Kionex)	15–30 g in 15–30 mL (70% sorbitol orally)	>2 hrs	4–6 hrs	Variable efficacy intestinal necrosis
Hemodialysis		Immediate	3 hrs	Arrhythmias (?)

IV, intravenously.

Calcium Therapy



This will **NOT** lower the total body or serum potassium levels but will reduce the risk of arrhythmia

- *Calcium is indicated if there are any ECG changes or evidence of cardiac instability. Consider in any patient with a K greater than 7.0*
- Calcium gluconate: Give 10ml of a 10% solution over 10 mins
- Calcium chloride is often used (can lead to thrombophlebitis)
- Use calcium with care in patients on Digoxin (controversial) as there are historical reports of patient deterioration

Dialysis

Insulin and Dextrose are effective but temporary treatments. Dialysis is the gold standard for removing potassium. This can provide immediate and reliable removal. Only use 8.4% Bicarbonate in acidotic patients.

Calcium Resonium

- **'There is Dubious Evidence for the use of this Drug'**
  - The resin was invented in the 1950s for industrial purposes and then someone had the idea of putting it in patients colons to exchange cations and hence bind potassium (mostly absorbed in the colon).
  - **Scherr et al (NEJM)**: http://www.ncbi.nlm.nih.gov/pubmed/13747532
    - This paper did show a drop in K

        - In the study there were 32 patients
        - They had Binding Resin +/- a laxative,
        - Most patients had glucose, insulin, low K diet and bicarbonate therapy - **THERE WAS NO CONTROL GROUP**
        - In the end in this study 23 /30 patients had a decrease of at least 0.4 in their K value… This was cited as evidence of effectiveness However, there was no control, and a lot of HCO3 / Insulin was used
    - A second paper in the same journal: http://www.ncbi.nlm.nih.gov/pubmed/13700297 also studied the use of binding resins with only 10 patients in the study
- One small study (http://www.ncbi.nlm.nih.gov/pubmed/9773794) in 1998 showed no change in serum potassium concentration after a single dose of binding resin or placebo (both with and without a sorbitol additive)
- The authors concluded – "*Because single-dose resin-cathartic therapy produces no or only trivial reductions in serum potassium concentration, and because this therapy is unpleasant and occasionally is associated with serious complications, this study questions the wisdom of its use in the management of acute hyperkalaemic episodes*."

What medications could cause a high potassium?

- **Drugs that change potassium movement across cell membranes** *(e.g. ß blockers, Digoxin, Potassium-containing drugs)*
- **Drugs that reduce aldosterone** *(e.g. ACE inhibitors; Angiotensin II receptor blockers, NSAIDs, heparin, renal transplant drugs)*
- **Drugs that inhibit aldosterone binding to the mineralocorticoid receptors** *(e.g. Spironolactone, Trimethoprim)*

Some Further Resources

- **Podcast** - Update on HyperK Management
    - http://www.onthewards.org/pods/83-hyperkalaemia
- **Paper** - ECG unreliable
    - Montague et al Clin J Am Soc Nephrol 3: 324-330, 2008
- **Podcast** - EMCrit Podcast 32 – Treatment of Severe Hyperkalemia - http://emcrit.org/podcasts/hyperkalemia
- **Debate** -
    - http://media.blubrry.com/emcrit/p/traffic.libsyn.com/emcrit/EMCrit-Bonus-Kayexalate-Useless.mp3