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Hyperkalaemia – The Acute Management

Hyperkalaemia Algorithm

Aim

To provide advice on management of all patients with acute hyperkalaemia in Children's Health Ireland

Definition of terms

Hyperkalaemia is defined as a potassium level greater than 5.5 mmol/l in children and more than 6 mmol/l in newborns [1].

Target Patient Population

Patients between ages 0-18 years old in Children's Health Ireland. This guideline is not intended for the management of patients with hyperkalaemia due to chronic renal failure.

Target Users

This guideline is directed at health-care professionals engaged in the care of children at CHI.

Assessment

Hyperkalaemia is potentially life threatening and can result in cardiac arrhythmias and sudden death. Hence this medical emergency requires prompt treatment.

The causes of hyperkalaemia can be categorised into three groups listed below [1]:

- **Increased intake:**

  An extremely high intake of potassium can lead to significant hyperkalaemia and arrhythmia, for example, following intravenous or oral potassium, penicillin [2] or a blood transfusion [3].

- **Redistribution of potassium from the intracellular to extracellular compartment:**

  1- **Pseudohyperkalaemia** due to cell breakdown following venepuncture or capillary sampling is the most frequent reason for a raised serum potassium result in children. A repeat free-flowing sample should be performed immediately to ensure the true potassium concentration is normal[1,4].
  2- **Metabolic acidosis** results in the movement of hydrogen ions into the intracellular space in order to buffer the intravascular pH. To maintain electroneutrality, potassium moves out of the cell resulting in hyperkalaemia [1,4].
  3- Insulin promotes movement of potassium into the cells, therefore **insulin deficiency,** as in diabetic ketoacidosis, can lead to hyperkalaemia [1,4].
  4- **Tissue breakdown** can cause the release of potassium from the cells into the extracellular fluid. Clinical examples include **trauma** or **severe hypothermia**, causing **rhabdomyolysis** [1,4]. **Chemotherapy**, causing the breakdown of lymphoma cells and of white cells in high-count leukaemia, can lead to **tumour lysis syndrome** [1,4]**. Haemolysis** [1] can also cause hyperkalaemia**.**

5- **Strenuous exercise** can also cause release of potassium from cells [1].
6- **Medication** such as β-blockers, will block the β-adrenergic facilitation of potassium uptake by cells and result in hyperkalemia.

- **Decreased renal excretion:**

1- **Impaired kidney function** leads to a reduction in potassium excretion, usually associated with reduced urine production (oliguric/anuric renal failure) [1,4]
2- **Reduced arterial blood volume** causes a reduction in the delivery of fluid to the distal site, where potassium is excreted and can result in hyperkalaemia [1,4]
3- **The absence of aldosterone,** or resistance to its effect, causes a reduction in potassium and hydrogen excretion, with coexistent **hyponatraemia** and metabolic **acidosis**. (e.g., **congenital adrenal hyperplasia, aldosterone synthase deficiency, hyperkalaemic distal renal tubular acidosis (type IV)** [1,4]
4- **Medications** can reduce effect of aldosterone and cause hyperkalaemia **(e.g., potassium sparing diuretics, ACE-inhibitors, NSAIDS, cyclosporin, trimethoprim, co-trimoxazole etc)** [1,4] **Beta-blockers and digoxin** prevent intracellular buffering of potassium as well as reduce the effectiveness of beta-2-agonists and insulin-glucose.

Symptoms [1]

Hyperkalaemia can be asymptomatic or symptomatic -

- Respiratory depression
- Palpitations, Arrhythmia, Cardiac arrest
- Paraesthesia, flaccid paralysis
- Ileus

ECG signs[1]

The following are the characteristic ECG changes with increasing levels of serum potassium [1]. **(Figure 1)**

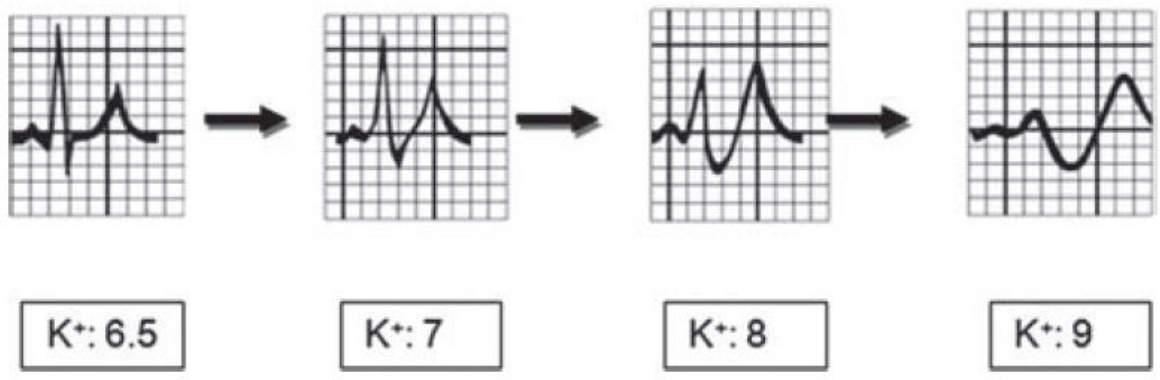

Figure 1 Changes in ECG with increasing hyperkalaemia. Serum K values are mmol/l.

1. Serum K+ 5.5–6.5 mmol/l – Tall, peaked T waves with narrow base.
2. ▸ Serum K+ 6.5–8.0 mmol/l – Peaked T waves, prolonged PR interval, decreased or disappearing P wave, widening of QRS, amplified R wave.
3. ▸ Serum K+ greater than 8.0 mmol/l – Changes occur due to delayed depolarisation. Absence of P wave, bundle branch blocks, progressive widening of QRS complex eventually

merging with the T wave to form the sine wave pattern. This is followed by ventricular fibrillation or asystole. Cardiac toxicity is enhanced by hypocalcaemia, hyponatraemia, metabolic acidosis and an acute rather than chronic rise in potassium [1].

Diagnosis and investigations

A full history can help identify the cause of the hyperkalaemia, it should include details on recent infections, bleeding, decreased oral intake, nausea and vomiting, diarrhoea, diet for potassium rich foods such as fruits, potatoes, beans and grains), medication, episodic weakness, failure to thrive, polydipsia and polyuria [1].

An ECG is mandatory but may be normal in significant hyperkalaemia and should not be used to guide management [1].

Management

- ***Hyperkalaemia is a medical emergency – seek consultant advice.***
- *Treatment is recommended when ECG changes are present or when serum potassium levels are greater than 6.0–6.5 mmol/L, regardless of the ECG [6].*
- *Immediately **stop all sources** of oral or parenteral potassium intake or medication that increases potassium concentration (*For example potassium sparing diuretics, ACE-inhibitors, NSAIDS and cyclosporin, etc)
- *Cardiac monitoring and frequent 12-lead ECG if abnormalities are present is mandatory.*

Calcium gluconate 10%w/v should be used first line in all patients with ECG changes. Repeat dose if ECG does not improve.

Management Principles

Stabilise the myocardium:

Calcium Gluconate infusion

Hyperkalaemia causes a decrease in membrane resting potential by inactivation of sodium channels and increasing membrane excitability. Calcium antagonises the effects of potassium and increases the resting membrane potential nearer to normal values [7]. It does not alter the potassium level but helps reduce excitability of the myocardium. The effects only last for 30–60 min, and so more definitive methods to lower serum potassium are required [1].

Dose:

Dose: 0.11 mmol/kg (0.5 ml/kg of Calcium Gluconate 10%w/v). Maximum 4.5mmol (20mL of Calcium Gluconate 10%w/v). By slow intravenous injection over 5–10 min: 0.11 mmol/kg (0.5 ml/kg) of calcium gluconate.
Dose may be repeated after 5 min if ECG changes persist. [8] See Calcium Gluconate 10%w/v Monograph in CHI Formulary for administration guidelines.

Note: 0.225mmol Calcium/1mL Calcium Gluconate 10%w/v
4.5mmol Calcium /20mL Calcium Gluconate 10%w/v

Driving extracellular potassium into the cell:

Insulin and Glucose Infusion

Insulin lowers the serum potassium by driving the potassium intracellularly in exchange for sodium. This is mediated via
the sodium potassium ATPase pump [1,5].

Dose:

By slow intravenous injection over 5-10 mins: Soluble Insulin (Actrapid®) 0.1units/Kg (Max.10units) + Glucose 0.5g/Kg IV over 5 mins mixed in the same syringe. Ideally given by CVC, if there is no central line, then a large peripheral vein may be used. See Insulin Monograph in CHI Formulary for stepwise preparation instructions.

Note glucose 50%w/v = glucose 0.5g/mL

Children >50Kg Prepare initial 5units (0.05mL) soluble Insulin (Actrapid®) plus 50mL Glucose 50%w/v in a 50mL syringe, then prepare remainder of dose in a second syringe.

Sodium bicarbonate infusion

Metabolic acidosis causes hydrogen ions to move intracellularly to maintain the intravascular pH as close to normal as possible, leading to an extracellular move of potassium in exchange to maintain electroneutrality. This movement is reversed if acidosis is corrected and will result in potassium moving into cell and reduce serum potassium [7 9].

Dose:

Intravenous sodium bicarbonate 8.4%w/v 1 mmol/kg (1mL/Kg) over 10–15 min. The onset of action is within 1 hour and the effects last for up to 2 hours. Potential complications include hypernatraemia, volume overload and tetany in patients with CKD and coexistent hypocalcaemia [8, 9, 10]. See Sodium Bicarbonate Monograph in CHI Formulary for administration guidelines.

β-2 Adrenergic agonists

β-2 Adrenergic agonists act by driving the potassium intracellularly by increasing sodium potassium ATPase activity.
Salbutamol is the most commonly used drug in this group, either nebulised or by intravenous infusions. Salbutamol can
produce a reduction in potassium of 0.5–1 mmol/l after nebulisation and 0.9–1.5 mmol/l after intravenous administration [7, 11].

Dose:

Nebulised salbutamol: 2.5 mg (under 5yrs age) or 5 mg (over 5yrs age). Doses may be repeated as needed every 2 hours[14].

Intravenous salbutamol: 4micrograms/kg given as an intravenous bolus over 5 min. Doses may be repeated as often as needed. The onset of action is rapid with effects lasting for up to 2 hours. Tachycardia is the main side effect and may be more pronounced than nebulised administration. [8] Intravenous salbutamol has not been shown to be more effective than nebulised salbutamol but may have more adverse related effects [11,12].

See Furosemide Monograph in CHI Formulary for dosing.

Discuss IV Salbutamol with a consultant prior to prescribing.

Removing excess potassium from the body

Diuretics

Loop diuretics prevent reabsorption of sodium and potassium in the loop of Henle and directly increase urinary potassium
excretion [1].

Dose:
Furosemide 1 mg/kg intravenously over at least 5 min. Usual max dose 40mg. Avoid rapid intravenous administration [8].
Ensure normal Renal function. See Furosemide Monograph in CHI Formulary for dosing.

Peritoneal dialysis, haemodialysis or continuous veno-venous haemofiltration

Renal replacement therapy is used when conservative methods fail. Haemodialysis (or continuous veno-venous haemofiltration in haemodynamically unstable patients) is more effective compared to peritoneal dialysis. A reduction in serum potassium level is seen immediately and the action is sustained as long as dialysis continues [1, 5, 7].

Treatment approach [13] : (Click for algorithm on Management of Hyperkalaemia in Children)

Mild Hyperkalaemia (Potassium <6mmol/L, asymptomatic and no ECG changes):

1. *Consider no treatment – Monitor*
2. Stop potassium supplements
3. Consider holding non-essential medications that can cause a rise in potassium.
4. Recheck result – if remains high
5. Salbutamol nebulised if no cardiac contra-indications

Moderate Hyperkalaemia (Potassium 6-7mmol/L, normal ECG, and asymptomatic):

1. Calcium gluconate 10%w/v to protect cardiac myocytes
2. Salbutamol nebulised if no contraindications
3. Insulin in glucose 50%w/v (See Formulary Administration Section for precise steps to prepare)
4. Sodium bicarbonate 8.4%w/v IV if metabolic acidosis

- Discuss with Consultant

Severe Hyperkalaemia (Potassium >7mmol/L, or symptomatic/ ECG changes):

1. Medical Emergency – Contact PICU for review immediately
2. Administer calcium gluconate 10%w/v to protect cardiac myocytes
3. Administer salbutamol nebulised (if no cardiac contraindications)
4. Administer insulin in glucose 50%w/v immediately

- Discuss with Consultant
- Consider hydrocortisone IV if suspect adrenal insufficiency
- Consider Dialysis

Medications Overview [12]

Medication	Response	Onset of Action	Duration of Action	Mechanism of Action	Expected decrease in potassium level	Considerations
Calcium gluconate 10%w/v	rapid	1-2mins	30-60mins	Protects cardiac myocytes	N/A	First line if ECG changes
Insulin in Glucose 50%w/v To be prepared & given together as per steps in formulary	intermediate	10-20mins	2-6hours	Shift potassium intracellularly	0.5-1.5mmol/L	Monitor for hyperglycaemia, consider insulin alone if hyperglycaemia
Salbutamol (Beta Agonist)	Intermediate	3-5mins	1-4hours	Shift potassium intracellularly	0.5-1.5mmol/L	IV Salbutamol may be considered, only after discussion with consultant. IV Salbutamol has **not** been shown to have a more effective or rapid effect on potassium, but has considerable side effects particularly cardiac (tachycardia).
Sodium Bicarbonate IV	Intermediate	30-60mins	2-6hours	Shift potassium intracellularly (questionable effect)	0.5mmol/L	(Only in metabolic acidosis patients)
Furosemide	delayed	5-30mins	2-6hours	Eliminates potassium from body	n/a	Caution if AKI suspected

Companion Documents

See CHI Formulary for most up to date dosing and administration guidelines available at http://olchcnet.hse.ie/CHI_at_Crumlin_Paediatric_Formulary/

CHI Smart Pump Drug Library available at http://olchcnet.hse.ie/Medicines_Information_/Standard_Concentration_Infusions/

Signature Sheet

Links to useful websites

- Clinical Practice Guidelines, The Royal Children's Hospital, Melbourne https://www.rch.org.au/clinicalguide/guideline_index/Hyperkalaemia/
- Medscape: Paediatric Hyperkalaemia Treatment and Management https://emedicine.medscape.com/article/907543-treatment

Link to Reference List