Hyperkalaemia - Management of –
Northern Beaches HS

Summary
Hyperkalaemia is a common but potentially life-threatening metabolic problem commonly encountered in hospitalised patients.

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Endorsed By
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Active

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Title: Management of Hyperkalaemia - NBHS

1. Preamble
Hyperkalaemia is a common but potentially life-threatening metabolic problem commonly encountered in hospitalised patients.

2. Scope of Practice
Hyperkalaemia will be managed according to directions of medical team. Responsibility of administration of medications will be shared by Nurses with guidance by Pharmacist.

3. Guideline
Hyperkalaemia may be defined as a serum potassium (K⁺) greater than 5.0 mmol/L.

3.1 Causes of Hyperkalaemia:
- Artifactual (sampling errors)
- Excessive intake
- Severe tissue damage
- Decreased excretion [caused by renal failure or by drugs (See table1)]
- Body fluid compartment shift (for example, acidosis)

3.2 Clinical features of Hyperkalaemia
- Tingling
- Paraesthesia
- Weakness
- Hypotension
- Bradycardia
- ECG changes

3.2.1 ECG changes: produce the classic electrocardiographic (ECG) manifestations of hyperkalaemia including (in order of their usual appearance):
- **Serum K⁺: 5.5 – 6.5 mmol/L**
  - Peaked T waves
  - Prolonged PR interval
- **Serum K⁺ 6.5 - 8.0 mmol/L**
  - Loss of the P wave
  - ST-segment elevation
  - Ectopic beats
  - Escape rhythm
- **Serum K⁺ > 8.0 mmol/L**
  - Progressive widening of QRS complex
  - Ventricular fibrillation
  - “Sine wave” configuration
  - Asystole
  - Axis deviations
  - Bundle branch blocks
  - Fascicular blocks

3.3.2. Neuromuscular Effects: Hyperkalaemia may result in paraesthesias and weakness progressing to a flaccid paralysis, which typically spares the diaphragm. Deep tendon reflexes are depressed or absent. Cranial nerves are rarely involved and sensory changes are minimal.

3.3.3. Metabolic Effects: Hyperkalaemia decreases renal ammoniagenesis which can produce a mild hyperchloreaemic metabolic acidosis and will limit the kidney’s ability to excrete an acid load and, thus, prevent correction of a metabolic acidosis.

3.4 Management:
In general, the initial treatment is independent of the cause of the disturbance, whereas the rational therapy of chronic hyperkalaemia depends on pathogenesis.
In considering when hyperkalaemia constitutes an emergency, several points should be considered. Firstly, the electrophysiologic effects of hyperkalaemia are directly proportional to both the absolute serum levels as well as its rate of rise. Secondly, concurrent metabolic disturbances may ameliorate (e.g., hypernatraemia, hypercalcaemia, alkalaemia) or exacerbate (e.g., hyponatraemia, hypocalcaemia, acidemia) the electrophysiologic consequences of hyperkalaemia. Lastly, although the ECG manifestations of hyperkalaemia are generally progressive and proportional to the serum levels, ventricular fibrillation may be the first ECG manifestation of hyperkalaemia; conversely, a normal ECG may be seen even with extreme hyperkalaemia.

Because most patients manifest hyperkalaemic ECG changes at serum $K^+ > 6.7$ mmol/L, it should be treated as an emergency if:

1) Serum $K^+ \geq 6.5$ mmol/L
2) ECG manifestations of hyperkalaemia are observed

The aim of therapy should be to:
1) Antagonize the effect of $K^+$ on excitable cell membranes.
2) Redistribute extracellular $K^+$ into cells.
3) Enhance elimination of $K^+$ from the body.

This guideline recommends that the algorithm in Figure 1 be used in management of hyperkalaemia. The standard regimen to antagonise the effects of $K^+$ on cardiac depolarization is a three-pronged approach of redistribution into the cell with: bicarbonate and insulin-dextrose infusion; enhancement of elimination by dialysis or use of Resonium A powder; and stabilisation of cardiac cell membranes with calcium gluconate infusion that can be given via a peripheral vein as long as one is careful not to allow extravasation. The dosages and administration of these therapeutic approaches should be in accordance with recommendations in table 2.

3.5 Monitoring:
1) ECG monitoring is required for all patients exhibiting ECG changes at baseline
2) pH should be checked to rule out acidosis
3) Bloods including:
   - Serum and urine creatinine level
   - Serum and urine osmolality
   - Serum and urine potassium
   - Serum calcium
   - Serum magnesium
   - Glucose
   - Electrolytes
4) Calculate Fractional Excretion of Potassium (FEK):

\[
\text{FEK} = \frac{\text{Urine Potassium / Serum Potassium}}{\text{Urine Creatinine / Serum Creatinine}} \times 100\% \\
\text{Interpretation: FEK < 10\% indicates renal aetiology and > 10\% indicates extra-renal cause.}
\]

5) Calculate Trans-Tubular Potassium Gradient ("TTKG"):

\[
\text{TTKG} = \frac{(U_{K^+}/P_{K^+}) + (U_{Osm}/P_{Osm})}{< 6 \Rightarrow \text{Aldosterone deficiency or resistance}} \]
\[
\text{> 10 \Rightarrow \text{Extra-renal cause hyperkalaemia}} 
\]
3.6 Follow-up:
Identify underlying cause of hyperkalaemia and treat if possible.

1) Dialysis for renal failure
2) Exogenous Fludrocortisone in mineralocorticoid deficiency
3) **Review medications** including potassium-sparing diuretics, ACE inhibitors / ARBs, potassium supplements, etc
4) **Dietician review** for advice on strict low potassium diet is essential

Hyperkalaemia caused by the use of ACE inhibitors or Angiotensin receptor blockers (ARBs) in patients with chronic kidney disease and metabolic acidosis may respond to sodium bicarbonate supplementation. The dosage is sodium bicarbonate (Sodibic) 840-1680mg (1-2 capsules) two to three times daily. Concomitant diuretic use reduces the risk of volume overload.

Table 1: Medications associated with hyperkalaemia.

<table>
<thead>
<tr>
<th>MEDICATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amiloride (Midamor)</td>
</tr>
<tr>
<td>Amiloride / Triamterene (Moduretic)</td>
</tr>
<tr>
<td>Amino acids</td>
</tr>
<tr>
<td>Angiotensin Receptor Blockers (ARBs)</td>
</tr>
<tr>
<td>Angiotensin Converting Enzyme (ACE) Inhibitors</td>
</tr>
<tr>
<td>Azole antifungals</td>
</tr>
<tr>
<td>Beta-blockers</td>
</tr>
<tr>
<td>Cyclosporine (Neoral)</td>
</tr>
<tr>
<td>Digoxin (at toxic levels)</td>
</tr>
<tr>
<td>Eplerenone (Inspra)</td>
</tr>
<tr>
<td>Ethinyl estradiol / drospirenone (Yasmin)</td>
</tr>
<tr>
<td>Fluoride toxicity</td>
</tr>
</tbody>
</table>
Figure 1: Recommended algorithm for management of hyperkalaemia

Is potassium elevated?

No

Yes

Is elevation real?

No

Yes

Is $K^+$ > 6.5mmol/L or Are there ECG changes present?

No

Patient requires emergency potassium reduction

ECG Abnormal

Yes

Give IV Calcium Gluconate

If acidotic: Administer IV Sodium Bicarbonate

Give Insulin-Dextrose infusion and/or Salbutamol by nebuliser

Order tests: Urinary potassium, creatinine & osmolality + serum potassium, creatinine & osmolality

Is $K^+$ < 6.5mmol/L?

No

Repeat Insulin-Dextrose infusion. Consider haemodialysis

Yes

Give Resonium A or Frusemide

Further evaluation and Long Term Therapy

Review medications: withhold or cease offending medication(s) immediately
### Table 2: Therapeutic options in the management of hyperkalaemia

<table>
<thead>
<tr>
<th>Agent</th>
<th>Dose</th>
<th>Onset of action</th>
<th>Duration of action</th>
<th>Complications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calcium Gluconate (10%)</td>
<td>10-20mL IV over 10 minutes</td>
<td>Immediate</td>
<td>30–60 min</td>
<td>Hypercalcaemia&lt;br&gt;Can worsen digoxin toxicity</td>
</tr>
<tr>
<td>Insulin-Dextrose infusion: regular Insulin</td>
<td>10 units with 50mL of 50% Dextrose as an IV push for 10 -15 minutes</td>
<td>20 min</td>
<td>4–6 hrs</td>
<td>Hypoglycaemia, consider 5% dextrose infusion at 100mL/hour especially when repeated doses required. Not necessary if BSL &gt; 13.9 mmol/L</td>
</tr>
<tr>
<td>(short-acting) “Actrapid”</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Salbutamol</td>
<td>20 mg in 4 mL normal saline solution nebulised over 10 minutes</td>
<td>30 min</td>
<td>2 hrs</td>
<td>Tachycardia (has never been studied in cardiac patients, caution advised)&lt;br&gt;Inconsistent response&lt;br&gt;May cause a brief initial rise in serum K⁺</td>
</tr>
<tr>
<td>Loop diuretics</td>
<td>Frusemide 40–80 mg IV</td>
<td>15 min</td>
<td>2–3 hrs</td>
<td>Volume depletion (give with saline if concerned)</td>
</tr>
<tr>
<td>Sodium bicarbonate</td>
<td>150 mmol/L IV at variable rate</td>
<td>Hours</td>
<td>Duration of infusion</td>
<td>Metabolic alkalosis&lt;br&gt;Volume overload</td>
</tr>
<tr>
<td>Sodium Polystyrene Sulfonate (Resonium A)</td>
<td>15–30 g in 15–30 mL water or Sorbitol</td>
<td>2 hrs (rectal route faster)</td>
<td>4–6 hrs</td>
<td>Variable efficacy, can cause Intestinal Necrosis, May lead to sodium retention Following enema colonic irrigation with a non-Na⁺, non-K⁺ containing solution e.g. 5% dextrose is advised to ensure adequate removal of the resin.</td>
</tr>
<tr>
<td>Haemodialysis</td>
<td></td>
<td>Immediate</td>
<td>3 hrs</td>
<td>Arrhythmias (?)&lt;br&gt;The excess total body potassium must be removed by either the intrinsic renal function or dialysis. Do not delay – other methods of control are temporary.</td>
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4. References


5. Revision & Approval History

<table>
<thead>
<tr>
<th>Date</th>
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