E03: Hyperkalemia

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Introduction

Although there are many potential electrolyte disturbances, hyperkalemia is arguably the most serious. In addition, it may be reasonably identified and treated in the out-of-hospital environment based on clinical features. The strict laboratory testing diagnosis of hyperkalemia is a serum potassium level over 5.5 mmol/L.

In rare cases with signs of hemodynamic compromise and potentially life-threatening arrhythmias, a clinical suspicion of hyperkalemia may be sufficient for initiating treatment.

Essentials

- The lethality of hyperkalemia is directly related to the rapidity with which the condition has developed, in addition to the absolute level of serum potassium.
- · Correlation of specific ECG changes with specific serum levels has not been adequately demonstrated.
- Clinical suspicion of hyperkalemia alone is not cause for treatment in the out-of-hospital setting.
- Treatment of life-threatening hyperkalemia aims at preventing or resolving lethal arrhythmias and restoring hemodynamic stability. This can be accomplished by stabilizing the myocardium, shifting potassium back into the intracellular space, and removing excess potassium from the body. The majority of these interventions are only available in hospital.

Additional Treatment Information

- Bradyarrhythmias with bizarre morphologies should prompt a strong consideration of hyperkalemia.
- To warrant out-of-hospital intervention, patients must present with significant hemodynamic or arrhythmogenic instability, alongside a suspicion of hyperkalemia as the likely cause.
- · Sodium Bicarbonate should only be used with a suspicion of concurrent underlying metabolic acidosis

General Information

- · Classic causes of hyperkalemia:
 - $\circ~$ Increased intake, either through potassium supplementation or diet
 - Increased production, as occurs in hemolysis, rhabdomyolysis, extensive burns, intense physical activity, or trauma (particularly crush injuries and tissue ischemia)
 - Decreased excretion, caused by acute or end-stage chronic renal failure, or by some drugs (such as nonsteroidal anti-inflammatory drugs, cyclosporine, potassium-sparing diuretics, and ACE inhibitors)
 - Shifts from intracellular to extracellular fluid as a result of acidosis (either metabolic or respiratory), insulin deficiency, or some drugs (particularly succinylcholine in certain populations, beta blockers, and digoxin)
- Clinical features of hyperkalemia are often non-specific:
 - $\circ~$ Generalized muscle weakness, paresthesia and/or absent deep tendon reflexes
 - In rare cases, muscular paralysis and hypoventilation may be observed
 - $\circ~$ Mental status change including confusion, fatigue, and lethargy
 - o Signs of renal failure, such as edema, skin changes, and dialysis sites, may be present
- The ECG is one of the most important diagnostic tools in detecting hyperkalemia. ECG changes associated with hyperkalemia include:
 - Tall, tented T-waves
 - Flattened or absent P-waves
 - Prolonged PR Interval
 - Wide QRS
 - · Bradycardia

• These changes may progress to bizarre QRS complexes, sine waves, or asystole.

Interventions

First Responder

- · Keep patient at rest in a position of comfort
- · Provide supplemental oxygen as required
 - → A07: Oxygen Administration

Emergency Medical Responder – All FR interventions, plus:

- Provide supplemental oxygen to maintain SpO₂ ≥ 94%
 - \bullet \rightarrow A07: Oxygen Administration
- Initiate rapid conveyance
- Consider intercept with additional resources

Primary Care Paramedic - All FR and EMR interventions, plus:

- Consider vascular access for hypotension or hypoperfusion
 - → D03: Vascular Access and Fluid Administration

Advanced Care Paramedic – All FR, EMR, and PCP interventions, plus:

- Obtain vascular access if not already done.
 - $\circ \rightarrow D03$: Vascular Access and Fluid Administration
- In patients with significant hemodynamic instability or dysrhythmia and a suspicion of hyperkalemia:
 - <u>CliniCall consultation required</u> prior to treatment of hyperkalemia.
 - Stabilize cellular action potential:
 - Calcium chloride
 - May repeat after 5 minutes if ECG changes persist or recur
 - Shift potassium intracellularly:
 - Sodium bicarbonate (only with a suspicion of concurrent underlying metabolic acidosis)
 - <u>Salbutamol</u>

Critical Care Paramedic – All FR, EMR, PCP, and ACP interventions, plus:

- Stabilize cellular action potential:
 - Calcium gluconate IV: 1.0 g slow push over 2-3 minutes; may repeat once after 5 minutes if ECG changes persist or recur
- Shift potassium intracellularly:
 - D10W with 10-20 U insulin R mixed: give 500 mL intravenously over 60 minutes, or:
 - Insulin R 10 units IV followed by glucose 25 g IV
 - Sodium bicarbonate IV: 150 mEq in 1 L D5W over 2-4 hours depending on volume status
- Eliminate potassium:
 - Furosemide IV: 40 mg every 12 hours
- Consider Kayexalate
 - o 30-60 g PO

References

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- 4. Porter RS et al. The Merck manual of diagnosis and therapy. 20th edition. 2018.