Guidelines

For The Management Of

Hyperkalemia

By

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Potassium is one of the body's major ions. Nearly 98% of the body's potassium is intracellular. The kidney determines potassium homeostasis, and excess potassium is excreted in the urine. The reference range for serum potassium level is 3.5-5 mEq/L, with total body potassium stores of approximately 50 mEq/kg (i.e., approximately 3500 mEq in a 70-kg person).

Hyperkalemia is a potentially life-threatening illness that can be difficult to diagnose because of a paucity of distinctive signs and symptoms.
.Hyperkalemia is defined as a potassium level greater than 5.5mEq/L:

- Mild 5.5-6.0 mEq/L
- Moderate 6.1-7.0 mEq/L
- Severe 7.0 mEq/L & greater **or**

if ECG changes or symptoms (muscle weakness or flaccid paralysis palpitations, paresthesias) occurring at ANY level of serum potassium ≥5.5mEql/L especially if associated with hypoxia

Causes

- *Decreased or impaired potassium excretion* - acute or chronic renal failure (most common), Hyperkalaemic renal tubular acidosis (type IV),potassium-sparing diuretics, urinary obstruction, sickle cell disease, Addison disease, and systemic lupus erythematosus
- *Additions of potassium into extracellular space* - potassium supplements (e.g. PO/IV potassium, salt substitutes), rhabdomyolysis, and hemolysis (e.g. blood transfusions), burns, tumor lysis syndrome
- *Transmembrane shifts*
  Hyperkalemic familial periodic paralysis, acidosis, insulin deficiency, catabolic states and medication effects (acute digitalis toxicity, beta-blockers, succinylcholine*)*
- *Drugs that*
  - Interfere with potassium excretion, e.g. amiloride, spironolactone
  - Interfere with the renin-angiotensin axis, e.g. angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARBs), non-steroidal anti-inflammatory drugs (NSAIDS), heparin.
- *Factitious or pseudohyperkalemia* - improper blood collection (e.g. ischemic blood draw from venipuncture technique), laboratory error, leukocytosis, and thrombocytosis
  - Prolonged tourniquet time.
  - There may have been difficulty collecting the sample.
  - The fist may have been clenched.
  - Test tube haemolysis, e.g. blood may have been squirted through a needle into the bottle or shaking the tube.
  - Use of the wrong anticoagulant, especially potassium EDTA.
  - Excessive cooling of specimen (in cold winter months)
  - Length of storage of the specimen.
  - Sample from limb receiving IV fluids containing potassium
  - Marked leukocytosis and thrombocytosis.

If there is doubt about the validity of the result, repeat the test

The history is most valuable in identifying conditions that may predispose to hyperkalemia. Cardiac and neurologic symptoms predominate: generalized fatigue, weakness, paresthesias, paralysis, palpitations

Physical examination

Evaluation of vital signs is essential to determine hemodynamic stability and presence of cardiac arrhythmias related to the hyperkalemia.

Cardiac examination may reveal extrasystoles, pauses or bradycardia.

Tachypnoea from respiratory muscle weakness

Neurologic examination may reveal diminished deep tendon reflexes or decreased motor strength.

In rare cases, muscular paralysis and hypoventilation may be observed.

Search for the stigmata of renal failure, such as edema, skin changes, and dialysis sites.

Look for signs of trauma that could put the patient at risk for rhabdomyolysis.

Laboratory Studies

Potassium level

BUN, creatinine ,glucose , calcium level

Arterial blood gas

Urine analysis

Cortisol & aldosterone level

ECG: ECG changes have a sequential progression of effects, which roughly correlate with the potassium level, though it is mandatory but it doesn't substitute serum potassium level

- ECG may be normal even in severe hyperkalemia.
- The absence of ECG changes does not mean: no need for treatment.
- Presence of ECG changes means need for urgent treatment.
- Potentially life-threatening arrhythmias can occur without warning at almost any level of hyperkalemia

Early changes of hyperkalemia include peaked T waves, shortened QT interval, and ST-segment depression, These changes are followed by bundle-branch blocks causing a widening of the QRS complex, increases in the PR interval, and decreased amplitude of the P wave, These changes reverse with appropriate treatment

Without treatment, the P wave eventually disappears and the QRS morphology widens to resemble a sine wave. Ventricular fibrillation or asystole follows.

Goals of treatment

1-stabilizing the myocardium

2- shifting potassium from the extracellular environment to the intracellular compartment

3-promoting the renal excretion and gastrointestinal loss of potassium

Assessment of the Patient

Is this "True or factitious Hyperkalemia?

How severe is the Hyperkalemia? Is urgent Treatment Required?

Why has the Patient got Hyperkalemia?

Management

Potassium ≥7.0 mmol/L, or any rise in potassium associated with ECG changes or symptoms, need to be **urgently** treated, begin treatment before diagnostic investigation of the underlying cause.

Individualize treatment based upon the patient's presentation, potassium level, and ECG

For mild elevations (5 - 6 mEq/L:)
dietary and medication changes, oral furosemide ,
Sodium polystyrene sulfonate (Kayexalate) orally or by enema
occasionally Fludrocortisone (florinef)can be given to outpatients

For moderate elevations (6 to 7 mEq/L)
shifting potassium intracellularly by
-Glucose plus insulin
-Nebulized albuterol (avoid if there is tachycardia or coronary artery disease)
- furosemide &/or kayexalate
-Sodium bicarbonate(unlikely to be effective without metabolic acidosis)
severe elevations (> 7 mEq/L with/without toxic ECG changes)
Intravenous access with frequent vital sign checks and continuous ECG monitoring
In the presence of hypotension or marked QRS widening, intravenous calcium, glucose and insulin, inhaled Albuterol, furosemide , kayexalat, sodium bicarbonate.
Be cautious in infusing calcium in cases of hyperkalemia associated digitalis toxicity. Magnesium sulfate (2 g over 5 min) may be used alternatively in the face of digoxin-toxic cardiac arrhythmias.
Calcium chloride or calcium gluconate: will not lower the potassium but are the first-line treatments for severe hyperkalemia when the ECG shows significant abnormalities
(e.g. widening of QRS interval, loss of P wave, cardiac arrhythmias).
One ampoule of calcium chloride has approximately 3 times more calcium than calcium gluconate but it is more arrhythmogenic
Doses should be titrated with constant monitoring of ECG changes during administration;
- Give 10 ml 10% calcium gluconate (1000 mg) which will improve ECG changes within 1-3 minutes, but this effect only has a transient effect 30-60 minutes.
- If no improvement then give 10 ml every 10 minutes till ECG normalizes (may need up to 50 ml).
- If patient is on Digoxin, **senior consultation** is mandatory as calcium should be infused much more slowly (add to 100 ml glucose 5%, run over an hour)and watch for Digoxin toxicity.
Dextrose with Insulin: Stimulates cellular uptake of K^+ within 20-30 min
- 10 U of regular insulin with one ampoule of D25 IV q 2-3 hrs or
10 units of regular insulin in 500 ml of 5% glucose infused over an hour
If the patient is diabetic & the serum glucose is ≥15mmol/L (270mg/dL)then administration of additional glucose with insulin is not required
Blood glucose needs to be checked before, during and after infusion hourly up to 6 hours after completion of the infusion as **delayed hypoglycaemia** is reported
Potassium will decrease (0.6-1.0 mEq /L) in 15 minutes and lasts 60 minutes.
Sodium bicarbonate: by increasing the pH, there will be temporary potassium shift from the extracellular to the intracellular environment.
Use 1-2 amps of 8.4% solution (50-100 mmol NaHCO3) IV over 10-20 minutes
Do not give with calcium, or it will precipitate.
Onset of action onset in 20 mins & lasts 2 hours
Only likely to be effective if underlying acidosis present.
Monitor blood pH to avoid excess alkalosis.

Beta2-adrenergic agonists: Albuterol (Ventolin)
It can be very beneficial in patients with renal failure when fluid overload is concern.
10 mg nebulised Salbutamol will reduces potassium (0.5-1.0 mEq /L) in 15-30 minutes and lasts 2hrs. The hypokalemic response is also weakened in patients taking beta-blockers and digoxin. Therefore salbutamol is not recommended as a single agent to treat hyperkalaemia

Diuretics: Furosemide
causes the loss of potassium through the kidney.
Effects are slow and frequently take an hour to begin.
40 to 80 mg IV Large doses may be needed in renal failure.

Binding resins: Sodium polystyrene sulfonate (Kayexalate)
Promote exchange of potassium for sodium in GI system.
15 gm in 20-100 mls water,sorbitol or glucose 1-4 times/day orally (works best) or
30-50 gms in 200 cc water 1-4 times/day as retention enema
Onset of action orally :2-12 h (longer when administered rectally), duration of action of 4-6 h.
Potassium level drops by approximately 0.5-1 mEq/L .Multiple doses usually necessary
Avoid if bowel obstruction. Watch for sodium overload

Dialysis
It is strongly considered if hyperkalemia is severe (level ≥7.0mmol/L) and other first-line agents have been unsuccessful, or if there is ongoing tissue damage and continued release of intracellular potassium is expected (burn, crush injury)

Further Outpatient Care
-Adjust diet to decrease potassium dietary load, avoid biscuits, chocolate coffee, potatoes fruit juice, fruits, fruit gums,.
-Adjust & substitute medications that predispose to or exacerbate hyperkalemia like
-KCL, potassium-sparing agents, beta blockers, ACEI/ARBs, NSAIDs
-Repeat potassium level tests in 2-3 days.
-Reevaluate renal function if signs of renal insufficiency are present.

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