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Hyperkalemia - a review article

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ABSTRACT

Introduction: Potassium is critical cation in physiological functions. Hyperkalemia is a common electrolyte disorder, defined as potassium level above 5,5 mmol/L. Knowledge of the basic rules of hyperkalemia management is crucial to every doctor.
Purpose: Systemic review of causes, symptoms and treatment of hyperkalemia.
Material and methods: Standard criteria were used to review the literature data. The search of articles in the PubMed database was carried out.
Description of the state of knowledge: The causes of hyperkalemia are different and mostly result from impaired urinary potassium excretion due to acute or chronic kidney disease. The symptoms of hyperkalemia vary and usually affect skeletal muscles, myocardium and central nervous system. Treatment of hyperkalemia depends on the presence or absence of symptoms, the severity of potassium elevation and the cause of hyperkalemia. Therefore, management with the patient with hyperkalemia includes careful interview, evaluation of clinical symptoms, ECG and searching for possible causes of disorders.
Summary: Hyperkalemia requires urgent therapeutic intervention. In each case of elevated potassium concentration ECG should be performed and the obtained laboratory result should be verified. The crucial point in hyperkalemia treatment is the shift of potassium to the cells and simultaneously the removal of potassium from the body.

Key words: potassium, hyperkalemia, ECG

1. Introduction

Potassium is primarily an intracellular cation, which is critical in physiological functions. The cells contain approximately 98 percent of body potassium. The largest amounts are found in skeletal muscles as well as in bones, liver or erythrocytes, reflecting potassium balance in the whole body. Therefore, measuring the concentration of potassium in the peripheral blood plasma is not a perfect measure and does not reflect body's resources. Potassium homeostasis depends on the coordinated interaction between tightly regulated potassium transfer in and out of the extracellular fluid compartment, and renal excretion or retention of potassium.

Regulation of potassium economy can be short- and long-term:

a) short-term:

- ✓ insulin - works on the Na+/K+-ATPase, which pumps Na+ out of and K+ into the cell in a 3:2 ratio
- ✓ metabolic acidosis - acidosis increases the plasma K^+ concentration by inducing a net shift of K^+ from the cellular to the extracellular compartment in exchange with H^+
- ✓ catecholamines (adrenaline, noradrenaline, dopamine) - stimulation of the sympathetic nervous system also moves the potassium into the cells (binding to beta-2 receptors on muscle cells stimulate the Na^+/K^+-ATPase and cause increased K^+ shift into cells)

b) long-term:

- ✓ renal function (passive and active mechanisms)

The correct concentration of potassium is 3,5-5 mmol/L. Hyperkalemia is a common electrolyte disorder, defined as potassium level above 5,5 mmol/L and can be classified according to serum potassium into mild (5,5–6,5 mmol/L), moderate (6,5–7,5 mmol/L)

and severe (>7.5 mmol/L) hyperkalemia. The causes of hyperkalemia are different and mostly result from impaired urinary potassium excretion due to acute or chronic kidney disease or the use of drugs that inhibit the renin-angiotensin-aldosterone system (RAAS). The treatment of hyperkalemia is aimed at inducing potassium loss.

Management with the patient with hyperkalemia includes careful interview, assessment of clinical symptoms, ECG and searching for possible causes of disorders. It should be always verified if the result of potassium concentration is reliable. When the patient with high levels of potassium does not have risk factors of hyperkalemia, pseudohyperkalemia should be considered in differential diagnosis. The following laboratory tests may be also helpful in diagnostics of hyperkalemia: creatinine, urea, calcium, glucose, acid base balance. [1, 2, 3, 4, 5, 6, 7, 8].

2. Causes of hyperkalemia

Hyperkalemia may result from many clinical disorders, connected with derangements of the homeostatic mechanisms that normally regulate potassium balance. The causes of hyperkalemia are listed below:

- medicines:
  - potassium
  - potassium-sparing diuretics (e.g., spironolactone)
  - NSAIDs
  - ACEi
  - beta-blockers
  - heparin
  - digoxin
  - trimethoprim
- impaired renal elimination of potassium
  - kidney failure
  - aldosterone deficiency
- impaired transport of potassium to the cells
  - blockade of beta-receptors (e.g., beta-blockers)
  - stimulation of alpha-receptors.
  - insulin deficiency
  - vegetative neuropathy
  - metabolic acidosis
- excessive release of potassium from the cells
  - rhabdomyolysis
  - state after hypothermia
  - tumor lysis syndrome
  - metabolic acidosis
  - sepsis
  - crush syndrome
  - extensive burns

The leading cause of hyperkalemia is acute and chronic kidney failure. Although, the etiology of hyperkalemia is often multifactorial, with impaired renal function, medication use, and hyperglycemia as the most common contributors. Because healthy individuals can adapt to excess potassium consumption by increasing excretion, increased potassium intake is rarely the sole cause of hyperkalemia, and underlying renal dysfunction is common [9, 10, 11, 12, 13, 14, 15, 16, 17, 18].

3. Pseudohyperkalemia

Pseudohyperkalemia is caused usually due to potassium movement out of the cells during or after the blood specimen has been drawn. The possible causes of pseudohyperkalemia may be connected with:

- technique of blood drawing (eg, mechanical trauma during venipuncture, repeated fist clenching)
- hemolysis
- high white blood cell count (>120,000/microL) (e.g., chronic lymphotic leukemia due to cell fragility)
- thrombocytosis (e.g., potassium moves out of platelets after clotting has occurred)

Pseudohyperkalemia should be always suspected when there is no apparent cause for hyperkalemia in an asymptomatic patient who has no clinical or ECG manifestations of elevated level of potassium [19, 20, 21].

4. Symptoms of hyperkalemia

The diagnosis of hyperkalemia is made by the detection of an elevated plasma or serum potassium level. Patient are often asymptomatic, the symptoms of hyperkalemia are uncharacteristic and may affect many organs:

a) skeletal muscles:

- ascending muscle weakness and reduced muscle strength
- periodic contractions of muscle groups
- paralysis of skeletal muscles
- epileptic seizures

b) myocardium:

- conduction abnormalities:
    - right bundle branch block
    - left bundle branch block
    - bifascicular block
    - advanced atrio-ventricular block
- cardiac arrhythmias:
    - sinus bradycardia
    - sinus arrest
    - slow idioventricular rhythms
    - ventricular tachycardia
    - ventricular fibrillation

- asystole
- decreased ejection volume of the left ventricle
- ECG changes (do not correlate with the serum potassium concentration):
  - tall peaked T waves
  - shortened QT interval
  - progressive leghthening of PR interval and QRS duration
  - disappearance of P wave
  - widening of QRS complex to a sine wave pattern

c) central nervous system:

- paresthesia
- tingling of the limbs
- entanglement
- metallic taste in the mouth

The functional consequences of hyperkalemia are assessed by the ECG monitoring and examination of muscle strength. Laboratory findings coexisting with hyperkalemia are usually: metabolic acidosis, hyponatremia and features of renal failure (e.g., high level of creatinine) [22, 23, 24, 25, 26, 27, 28, 29, 30].

5. Treatment of hyperkalemia

The goals of hyperkalemia treatment are following:

1) the protection of the myocardium:

- CALCIUM
  - usually calcium chloride (10 mL of a 10 percent solution) or calcium gluconate (also 10 mL of a 10 percent solution) intravenously
  - infused over two to three minutes with constant cardiac monitoring
  - the dose can be repeated after 5 minutes if the ECG changes persist or recur
  - calcium infusions may cause tissue necrosis – as a consequence central or deep vein administration of calcium is preferred
  - the goal: stabilisation of cell membranes (because hipocalcemia increases the cardiotoxicity of hyperkalemia)
  - contraindications: digoxin-intoxication, hypercalcemic states

2) the displacement of extracellular potassium into cells (shift from extra- to intracellular space):

- INSULIN WITH GLUCOSE
  - usually a bolus injection of 10 units of regular insulin given with 25 g of glucose to prevent the development of hypoglycemia
  - the serum glucose should be measured every hour for five to six hours after the administration of insulin because of the risk of hypoglycaemia
  - the goal: insulin drives potassium into cells by enhancing the activity of the Na-K-ATPase pump in skeletal muscle
  - the effect of insulin begins in 10 to 20 minutes and lasts for 4 to 6 hours
  - glucose is given with insulin to prevent hypoglycaemia, but if the serum glucose is ≥250 mg/dL (13.9 mmol/L) insulin should be given alone
  - side effects: hypoglycaemia
- BETA-2-ADRENERGIC AGONISTS (salbutamol, reproterol)

- also drive potassium into the cells via the Na-K-ATPase pump in skeletal muscle
- besides, beta-2-adrenergic receptors may activate the inwardly directed Na-K-2Cl cotransporter
- e.g., salbutamol via nebulizer or alternatively given intravenously
- side effects: tachycardia

- SODIUM BICARBONATE
  - for acidotic patients
  - goal: hydrogen ion release from the cells as part of the buffering reaction, accompanied by potassium movement into the cells to maintain electroneutrality
  - side effects: rise in pH can aggravate hypocalcemia
- LOOP DIURETICS
  - usually 40 mg of intravenous furosemide every 12 hours
  - goal: loop diuretics increase potassium loss in the urine, particularly when combined with saline hydratation to maintain distal sodium delivery and flow
  - side effects: electrolyte disorders, metabolic alkalosis, hypotension, hypersensitivity reactions, ototoxicity
- CATION EXCHANGERS

a) NEWER CATION EXCHANGERS – PATIROMER OR ZS-9

- nonabsorbable compounds that exchange calcium or sodium and hydrogen
- patiromer is a spherical organic polymer, formulated as a powder for suspension, which binds potassium in the colon in exchange for calcium
- zirconium cyclosilicate (ZS-9) is an inorganic crystalline compound that exchanges both sodium and hydrogen ions for potassium throughout its intestinal transit

b) SODIUM POLYSTYRENE SULFONATE (SPS)

- oral dosage usually 15 to 30 g every 6 hours
- goal: gastrointestinal cation exchange therapy
- criteria for using SPS: life-threatening hyperkalemia, dialysis and newer cation exchangers are not available, other therapies have failed
- side effects: intestinal necrosis (that is why SPS should be given with sorbitol)

- RENAL REPLACEMENT THERAPY (RRT) - HEMODIALYSIS
  - hemodialysis is faster than peritoneal dialysis and provides a substantially higher potassium clearance than continuous forms of RRT
  - hemodialysis is indicated in hyperkalemia with severe renal impairment

Marked electrocardiographic changes or severe muscle weakness may require immediate treatment. Therefore, treatment of hyperkalemia depends on the presence or absence of hyperkalemia symptoms, the severity of potassium elevation and the cause of hyperkalemia [31, 32, 33, 34, 35, 36, 37, 38, 39, 40, 41, 42, 43, 44, 45, 46, 47, 48, 49, 50, 51].

6. Prevention

Patients with chronic kidney disease should take steps to prevent hyperkalemia:

- avoiding episodes of fasting, which can increase potassium movement out of the cells due, at least in part, to reduced insulin
- avoiding drugs that raise the serum potassium concentration (e.g., angiotensin-converting enzyme (ACE) inhibitors, angiotensin II receptor blockers (ARBs), direct renin inhibitors, aldosterone antagonists, nonselective beta blockers)

- monitoring of the serum potassium concentration and estimated glomerular filtration rate (eGFR)
- low-potassium diet
- the use of thiazide or loop diuretics whenever otherwise indicated [52, 53, 54, 55].

7. Dietary restrictions

Maintenance of adequate potassium intake or the administration of potassium supplements usually lowers the blood pressure, particularly in blacks and in patients who are not sodium restricted. Furthermore, a higher potassium intake reduces the risk of stroke. The mechanism by which potassium reduces blood pressure is not clear. Some experts suggest that hypertensive patients should consume at least 120 mEq (4.7 g) of dietary potassium/day, provided they do not have a predisposition to hyperkalemia. This level of potassium intake can be achieved preferably with dietary counselling [56, 57, 58, 59, 60].

Patients usually need to be on a low-potassium diet to treat or prevent hyperkalemia. So what is a low-potassium diet?

1) choosing foods with low levels of potassium
2) avoiding foods with high levels of potassium (table 1)

Table 1

foods that are low in potassium	foods that are high of potassium
almonds	avocado
apple juice	bananas
asparagus	broccoli
blackberries	brussels sprouts
blueberries	cabbage
carrots (cooked)	carrots (raw)
celery	chocolate
cherries	clam
chicken	coconut
corn	figs
cranberries	kiwi
cucumber	mango
eggs	milk and dairy products
flax seed	olives
grapes	oranges
lettuce	potatoes
onions	pumpkin
peanuts	salmon
plumps	sardines
pineapple	soy milk
raspberries	sports drinks
spinach	steak
strawberries	tomatoes
turkey	whitefish
zucchini	yogurt

8. Summary

Due to serious consequences hyperkalemia requires urgent therapeutic intervention. Knowledge of the basic rules of hyperkalemia management should be obligatory to every doctor. In each case of elevated potassium concentration ECG should be performed and the obtained laboratory result should be verified. The crucial point in hyperkalemia treatment is the shift of potassium to the cells and simultaneously the removal of potassium from the body [61, 62].

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