What is hyperkalemia?

Hyperkalemia is a common diagnosis. Fortunately, most patients who are diagnosed have mild hyperkalemia (which is usually well tolerated). However, any condition causing even mild hyperkalemia should be treated to prevent progression into more severe hyperkalemia. Extremely high levels of potassium in the blood (severe hyperkalemia) can lead to cardiac arrest and death. When not recognized and treated properly, severe hyperkalemia results in a high mortality rate.

Technically, hyperkalemia means an abnormally elevated level of potassium in the blood. The normal potassium level in the blood is 3.5-5.0 milliequivalents per liter (mEq/L). Potassium levels between 5.1 mEq/L to 6.0 mEq/L reflect mild hyperkalemia. Potassium levels of 6.1 mEq/L to 7.0 mEq/L are moderate hyperkalemia, and levels above 7 mEq/L are severe hyperkalemia.

How does hyperkalemia affect the body?

Potassium is critical for the normal functioning of the muscles, heart, and nerves. It plays an important role in controlling activity of smooth muscle (such as the muscle found in the digestive tract) and skeletal muscle (muscles of the extremities and torso), as well as the muscles of the heart. It is also important for normal transmission of electrical signals throughout the nervous system within the body.

Normal blood levels of potassium are critical for maintaining normal heart electrical rhythm. Both low blood potassium levels (hypokalemia) and high blood potassium levels (hyperkalemia) can lead to abnormal heart rhythms.

The most important clinical effect of hyperkalemia is related to electrical rhythm of the heart. While mild hyperkalemia probably has a limited effect on the heart, moderate hyperkalemia can produce EKG changes (EKG is a reading of the electrical activity of the heart muscles), and severe hyperkalemia can cause suppression of electrical activity of the heart and can cause the heart to stop beating.

Another important effect of hyperkalemia is interference with functioning of the skeletal muscles. Hyperkalemic periodic paralysis is a rare inherited disorder in which patients can develop sudden onset of hyperkalemia which in turn causes muscle paralysis. The reason for the muscle paralysis is not clearly understood, but it is probably due to hyperkalemia suppressing the electrical activity of the muscle.

What are the symptoms of hyperkalemia?

Hyperkalemia can be asymptomatic, meaning that it causes no symptoms. Sometimes, patients with hyperkalemia report vague symptoms including:

- nausea,
- fatigue,
- muscle weakness, or
- tingling sensations.

More serious symptoms of hyperkalemia include slow heartbeat and weak pulse. Severe hyperkalemia can result in fatal cardiac standstill (heart stoppage). Generally, a slowly rising potassium level (such as with chronic kidney failure) is better tolerated than an abrupt rise in potassium levels. Unless the rise in potassium has been very rapid, symptoms of hyperkalemia are usually not apparent until potassium levels are very high (typically 7.0 mEq/l or higher).

Symptoms may also be present that reflect the underlying medical conditions that are causing the hyperkalemia.

What causes hyperkalemia?

The major causes of hyperkalemia are kidney dysfunction, diseases of the adrenal gland, potassium shifting out of cells into the blood circulation, and medications.

Hyperkalemia and kidney dysfunction

Potassium is normally excreted by the kidneys, so disorders that decrease the function of the kidneys can result in hyperkalemia. These include:

- acute and chronic renal failure,
• glomerulonephritis,
• lupus nephritis,
• transplant rejection, and
• obstructive diseases of the urinary tract, such as urolithiasis (stones in the urinary tract).

Furthermore, patients with kidney dysfunctions are especially sensitive to medications that can increase blood potassium levels. For example, patients with kidney dysfunctions can develop worsening hyperkalemia when given salt substitutes that contain potassium, potassium supplements (either orally or intravenously), or medications that can increase blood potassium levels. Examples of medications that can increase blood potassium levels include:

  • ACE inhibitors,
  • nonsteroidal anti-inflammatory drugs (NSAIDs),
  • Angiotensin II Receptor Blockers (ARBs), and
  • potassium-sparing diuretics (see below).

Diseases of the adrenal gland

Adrenal glands are small glands located adjacent to the kidneys, and are important in secreting hormones such as cortisol and aldosterone. Aldosterone causes the kidneys to retain sodium and fluid while excreting potassium in the urine. Therefore diseases of the adrenal gland, such as Addison's disease, that lead to decreased aldosterone secretion can decrease kidney excretion of potassium, resulting in body retention of potassium, and hence hyperkalemia.

Hyperkalemia & potassium shifts

Potassium can move out of and into cells. Our total body potassium stores are approximately 50 mEq/kg of body weight. At any given time, about 98% of the total potassium in the body is located inside of cells (intracellular), with only 2% located outside of cells (in the blood circulation and in the extracellular tissue). The blood tests for measurement of potassium levels measure only the potassium that is outside of the cells. Therefore, conditions that can cause potassium to move out of the cells into the blood circulation can increase the blood potassium levels even though the total amount of potassium in the body has not changed.

One example of potassium shift causing hyperkalemia is diabetic ketoacidosis. Insulin is vital to patients with type 1 diabetes. Without insulin, patients with type 1 diabetes can develop severely elevated blood glucose levels. Lack of insulin also causes the breakdown of fat cells, with the release of ketones into the blood, turning the blood acidic (hence the term ketoacidosis). The acidosis and high glucose levels in the blood work together to cause fluid and potassium to move out of the cells into the blood circulation. Patients with diabetes often also have diminished kidney capacity to excrete potassium into urine. The combination of potassium shift out of cells and diminished urine potassium excretion causes hyperkalemia.

Another cause of hyperkalemia is tissue destruction, dying cells release potassium into the blood circulation. Examples of tissue destruction causing hyperkalemia include:

  • trauma,
  • burns,
  • surgery,
  • hemolysis (disintegration of red blood cells),
  • massive lysis of tumor cells, and
  • rhabdomyolysis (a condition involving destruction of muscle cells that is sometimes associated with muscle injury, alcoholism, or drug abuse).

Hyperkalemia medications

Potassium supplements, salt substitutes that contain potassium and other medications can cause hyperkalemia.

In normal individuals, healthy kidneys can adapt to excessive oral intake of potassium by increasing urine excretion of potassium, thus preventing the development of hyperkalemia. However, taking in too much potassium (either through foods, supplements, or salt substitutes containing potassium) can cause hyperkalemia if there is kidney dysfunction or if the patient is taking medications that decrease urine potassium excretion such as ACE inhibitors and potassium-sparing diuretics.

Examples of medications that decrease urine potassium excretion include:

  • ACE inhibitors,
  • ARBs,
  • NSAIDs,
  • potassium-sparing diuretics such as:
    - spironolactone (Aldactone),
    - triamterene (Dyrenium), and
- trimethoprim-sulfamethoxazole (Bactrim).

Even though mild hyperkalemia is common with these medications, severe hyperkalemia usually does not occur unless these medications are given to patients with kidney dysfunction.

How is hyperkalemia diagnosed?

Blood is withdrawn from a vein (like other blood tests). The potassium concentration of the blood is determined in the laboratory. If hyperkalemia is suspected, an electrocardiogram (ECG or EKG) is often performed, since the ECG may show changes typical for hyperkalemia in moderate to severe cases. The ECG will also be able to identify cardiac arrhythmias that result from hyperkalemia.

How is hyperkalemia treated?

Treatment of hyperkalemia must be individualized based upon the underlying cause of the hyperkalemia, the severity of symptoms or appearance of ECG changes, and the overall health status of the patient. Mild hyperkalemia is usually treated without hospitalization especially if the patient is otherwise healthy, the ECG is normal, and there are no other associated conditions such as acidosis and worsening kidney function. Emergency treatment is necessary if hyperkalemia is severe and has caused changes in the ECG. Severe hyperkalemia is best treated in the hospital, oftentimes in the intensive care unit, under continuous heart rhythm monitoring.

Treatment of hyperkalemia may include any of the following measures, either singly or in combination:

- A diet low in potassium (for mild cases).
- Discontinue medications that increase blood potassium levels.
- Intravenous administration of glucose and insulin, which promotes movement of potassium from the extracellular space back into the cells.
- Intravenous calcium to temporarily protect the heart and muscles from the effects of hyperkalemia.
- Sodium bicarbonate administration to counteract acidosis and to promote movement of potassium from the extracellular space back into the cells.
- Diuretic administration to decrease the total potassium stores through increasing potassium excretion in the urine. It is important to note that most diuretics increase kidney excretion of potassium. Only the potassium-sparing diuretics mentioned above decrease kidney excretion of potassium.
- Medications that stimulate beta-2 adrenergic receptors, such as albuterol and epinephrine, have also been used to drive potassium back into cells.
- Medications known as cation-exchange resins, which bind potassium and lead to its excretion via the gastrointestinal tract.
- Dialysis, particularly if other measures have failed or if renal failure is present.

Treatment of hyperkalemia also includes treatment of any underlying causes (for example, kidney disease, adrenal disease, tissue destruction) of hyperkalemia.

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