Hypokalemia

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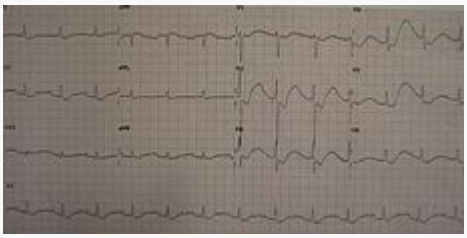
Hypokalemia	
Classification and external resources	
	
An ECG in a person with a potassium level of 1.1 showing the classical ECG changes of ST segment depression, inverted T waves, large U waves, and a slightly prolonged PR interval.	
ICD-10	E87.6
ICD-9	276.8
DiseasesDB	6445
MedlinePlus	000479
eMedicine	emerg/273
MeSH	D007008

Hypokalemia (American English) or **hypokalaemia** (British English), also **hypopotassemia** or **hypopotassaemia** (ICD-9), refers to the condition in which the concentration of potassium (K^+) in the blood is low. The prefix *hypo-* means "under" (contrast with *hyper-*, meaning "over"); *kal-* refers to*kalium*, the Neo-Latin for potassium, and *-emia* means "condition of the blood."

Normal plasma potassium levels are between 3.5 to 5.0 mEq/L;[1] at least 95% of the body's potassium is found inside cells, with the remainder in the blood. This concentration gradient is maintained principally by the Na+/K+ pump.

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[edit]Signs and symptoms

Mild hypokalemia is often without symptoms, although it may cause a small elevation of blood pressure,[2] and can occasionally provoke cardiac arrhythmias. Moderate hypokalemia, with serum potassium concentrations of 2.5-3 mEq/L (Nl: 3.5-5.0 mEq/L), may cause muscular weakness, myalgia, and muscle cramps (owing to disturbed function of the skeletal muscles), and constipation (from disturbed function of smooth muscles). With more severe hypokalemia, flaccid paralysis and hyporeflexia may result. There are reports of rhabdomyolysis occurring with profound hypokalemia with serum potassium levels less than 2 mEq/L. Respiratory depression from severe impairment of skeletal muscle function is found in many patients.

Some electrocardiographic (ECG) findings associated with hypokalemia include **flattened or inverted T waves**, a U wave, ST depression and a wide PR interval. Due to prolonged repolarization of ventricular Purkinje fibers, a prominent U wave occurs, that is frequently superimposed upon the T wave and therefore produces the appearance of a prolonged QT interval.[3]

[edit]Causes

Hypokalemia can result from one or more of the following medical conditions:

[edit]Inadequate potassium intake

- Perhaps the most obvious cause is insufficient consumption of potassium (that is, a low-potassium diet) or starvation. However, without excessive potassium loss from the body, this is a rare cause of hypokalemia.

[edit]Gastrointestinal/integument loss

- A more common cause is excessive loss of potassium, often associated with heavy fluid losses that "flush" potassium out of the body. Typically, this is a consequence of diarrhea, excessiveperspiration, or losses associated with surgical procedures. Vomiting can also cause hypokalemia, although not much potassium is lost from the vomitus. Rather, there are heavy urinary losses of K^+ in the setting of post-emetic bicarbonaturia that force urinary potassium excretion (see Alkalosis below). Other GI causes include pancreatic fistulae and the presence ofadenoma.

[edit]Urinary loss

- Certain medications can cause excess potassium loss in the urine. Diuretics, including thiazide diuretics (e.g. hydrochlorothiazide) and loop diuretics (e.g. furosemide) are a common cause of hypokalemia. Other medications such as the antifungal, amphotericin B, or the cancer drug, cisplatin, can also cause long-term hypokalemia.
- A special case of potassium loss occurs with diabetic ketoacidosis. In addition to urinary losses from polyuria and volume contraction, there is also obligate loss of potassium from kidney tubules as a cationic partner to the negatively charged ketone, β-hydroxybutyrate.
- Hypomagnesemia can cause hypokalemia. Magnesium is required for adequate processing of potassium. This may become evident when hypokalemia persists despite potassium supplementation. Other electrolyte abnormalities may also be present.
- Alkalosis can cause transient hypokalemia by two mechanisms. First, the alkalosis causes a shift of potassium from the plasma and interstitial fluids into cells; perhaps mediated by stimulation of Na^+-H^+ exchange and a subsequent activation of Na^+/K^+-ATPase activity.[4] Second, an acute rise of plasma HCO_3^- concentration (caused by vomiting, for example) will exceed the capacity of the renal proximal tubule to reabsorb this anion, and potassium will be excreted as an obligate cation partner to the bicarbonate.[5] Metabolic alkalosis is often present in states of volume depletion, so potassium is also lost via aldosterone-mediated mechanisms.
- Disease states that lead to abnormally high aldosterone levels can cause hypertension and excessive urinary losses of potassium. These include renal artery stenosis and tumors (generally non-malignant) of the adrenal glands, e.g., Conn's syndrome (primary hyperaldosteronism). Cushing's syndrome can also lead to hypokalemia due to excess cortisol binding the Na+/K+ pump and acting like aldosterone. Hypertension and hypokalemia can also be seen with

a deficiency of the 11-beta-hydroxysteroid dehydrogenase type 2 enzyme which allows cortisols to stimulate aldosterone receptors. This deficiency—known as apparent mineralocorticoid excess syndrome—can either be congenital or caused by consumption of glycyrrhizin, which is contained in extract of licorice, sometimes found in herbal supplements, candies and chewing tobacco.

- Rare hereditary defects of renal salt transporters, such as Bartter syndrome or Gitelman syndrome, can cause hypokalemia, in a manner similar to that of diuretics. As opposed to disease states of primary excesses of aldosterone, blood pressure is either normal or low in Bartter's or Gitelman's.

[edit]Distribution away from ECF

- In addition to alkalosis, other factors can cause transient shifting of potassium into cells, presumably by stimulation of the Na-K-ATPase.[4] These hormones and medications include insulin,epinephrine, and other beta agonists (e.g. salbutamol or salmeterol), and xanthines (e.g. Theophylline).[6]

- Rare hereditary defects of muscular ion channels and transporters that cause hypokalemic periodic paralysis can precipitate occasional attacks of severe hypokalemia and muscle weakness. These defects cause a heightened sensitivity to the normal changes in potassium produced by catechols and/or insulin and/or thyroid hormone, which lead to movement of potassium from the extracellular fluid into the muscle cells.

[edit]Other/ungrouped

- There have been a handful of published reports describing individuals with severe hypokalemia related to chronic extreme consumption (4-10 L/day) of colas.[7] The hypokalemia is thought to be from the combination of the diuretic effect of caffeine[8] and copious fluid intake, although it may also be related to diarrhea caused by heavy fructose ingestion.[9][10] A physiological response to Hypercapnia, blood potassium (as well as calcium) helps offset Acidosis, which is consistent with chronic, extreme consumption of carbonated beverages.

[edit]Pseudohypokalemia

- Pseudohypokalemia is a decrease in the amount of potassium that occurs due to excessive uptake of potassium by metabolically active cells in a blood sample after it has been drawn. It is a laboratory artifact that may occur when blood samples remain in warm conditions for several hours before processing.[11]

[edit]Pathophysiology

Potassium is essential for many body functions, including muscle and nerve activity. The electrochemical gradient of potassium between the intracellular and extracellular space is essential for nerve function; in particular, potassium is needed to repolarize the cell membrane to a resting state after an action potential has passed. Lower potassium levels in the extracellular space will cause hyperpolarization of the resting membrane potential. This hyperpolarization is caused by the effect of the altered potassium gradient on resting membrane potential as defined by theGoldman equation. As a result, a greater than normal stimulus is required for depolarization of the membrane in order to initiate an action potential.

In the heart, hypokalemia causes hyperpolarization in the myocytes' resting membrane potential. The more negative membrane potentials in the atrium may cause arrhythmias because of more complete recovery from sodium-channel inactivation, making the triggering of an action potential more likely. In addition, the reduced extracellular potassium (paradoxically) inhibits the activity of the I_{Kr} potassium current[12] and delays ventricular repolarization. This delayed repolarization may promote reentrant arrhythmias.

[edit]Treatment

The most important treatment in severe hypokalemia is addressing the cause, such as improving the diet, treating diarrhea or stopping an offending medication. Patients without a significant source of potassium loss and who show no symptoms of hypokalemia may not require treatment.

Mild hypokalemia (>3.0 mEq/L) may be treated with oral potassium chloride supplements (Klor-Con, Sando-K, Slow-K). As this is often part of a poor nutritional intake, potassium-containing foods may be recommended, such as leafy green vegetables, tomatoes, citrus fruits, oranges or bananas.[13] Both dietary and pharmaceutical supplements are used for people taking diuretic medications (see **Causes**, above).

Severe hypokalemia (<3.0 mEq/L) may require intravenous (IV) supplementation. Typically, a saline solution is used, with 20-40 mEq KCl per liter over 3–4 hours. Giving IV potassium at faster rates (20-25 mEq/hr) may predispose to ventricular tachycardias and requires intensive monitoring. A generally safe rate is 10 mEq/hr. Even in severe hypokalemia, oral supplementation is preferred given its safety profile. Sustained release formulations should be avoided in acute settings.

Difficult or resistant cases of hypokalemia may be amenable to a potassium-sparing diuretic, such as amiloride, triamterene, or spironolactone or eplerenone. Concomittant hypomagnesiumemia will inhibit potassium replacement as magnesium is a cofactor for potassium uptake.[14]

When replacing potassium intravenously, infusion via a central line is encouraged to avoid the frequent occurrence of a burning sensation at the site of a peripheral IV, or the rare occurrence of damage to the vein. When peripheral infusions are necessary, the burning can be reduced by diluting the potassium in larger amounts of IV fluid, or mixing 3 ml of 1% lidocaine to each 10 meq of KCl per 50 ml of IV fluid. The practice of adding lidocaine, however, raises the likelihood of serious medical errors.[15]

[edit]In other animals

[edit]Cats

Burmese hypokalaemia (Familial Episodic Hypokalaemic Polymyopathy) is characterised by episodes of low serum potassium levels and high CPK (an enzyme that indicates muscle damage). Clinical signs include skeletal muscle weakness, which is episodic in nature and can affect the whole animal or may be localised to certain muscles. This is most

obvious in the neck muscles, but sometimes occurs in just the limbs. As a result affected cats tend to have problems walking and holding their head correctly.

[edit]Genetic mutation causing Burmese hypokalaemia

A research team consisting of veterinarians and geneticists from the University of Bristol (Langford), UC Davis, University of Sydney, Massey University and Justus Liebig University have recently identified the genetic mutation responsible for Burmese hypokalaemia, allowing a genetic test to be developed (see here). This is an autosomal recessive disease, meaning that two copies of the mutated gene are required for disease.

[edit]Cat breeds at risk of Burmese hypokalaemia

- Asian
- Australian Mist
- Bombay
- Burmese
- Burmilla
- Cornish Rex
- Devon Rex
- Singapura
- Sphynx
- Tiffanie
- Tonkinese

[edit]Management of affected cats

Affected cats can usually be managed effectively by giving potassium supplements to their diet [16]. This will reduce the signs of disease or minimise their frequency and severity. In some affected cats signs seem to disappear when they get to 1-2 years of age without the need for further treatment.

A feline form of hypokalemic periodic paralysis has been described in Burmese kittens, which appears to be related to an autosomal recessive mutation. Although these kittens are not hypokalemic between episodes, regular supplementation of [KCl] seems effective.[17]

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