Hypokalemia

We define **hypokalemia as** K+ < 3.5 mmol/l (the lower and upper limits of potassium may vary slightly with respect to local laboratory reference limits). It can be a real potassium deficiency or a simple movement of extracellular potassium into the cells. The potassium values must always be corrected to the ABR values and the shape of the ECG curve.

The potassium values must always be related to the pH values and the shape of the ECG curve.

Etiopathogenesis

- **Transcellular movement of potassium:** acute changes in ABR affect the transcellular distribution of potassium. Therefore, both acute MAL and RAL are often accompanied by severe hypokalemia due to the movement of potassium into the cell in exchange for a hydrogen ion.
- **Inadequate potassium intake:** is a rare cause of hypokalemia today. It has been observed in infants receiving commercially produced low-chlorinated cow's milk; hypochloremic MAL and hypokalemia occurred.
- **Losses of potassium by the kidneys:** caused by the administration of diuretics , excessive activity of mineralocorticoids , RTA , hyperreninemia, diabetic ketoacidosis .
- **Potassium losses from the GIT:** vomiting and/or diarrhea.
- A rare cause of hypokalemia is hypokalemic periodic paralysis. It is a disease with AD heredity. The basis is a mutation of the α-1S subunit of the T-tubule calcium channel of the muscle cell or a mutation of the ryanodine receptor, which controls the release of calcium from the sarcoplasmic reticulum.

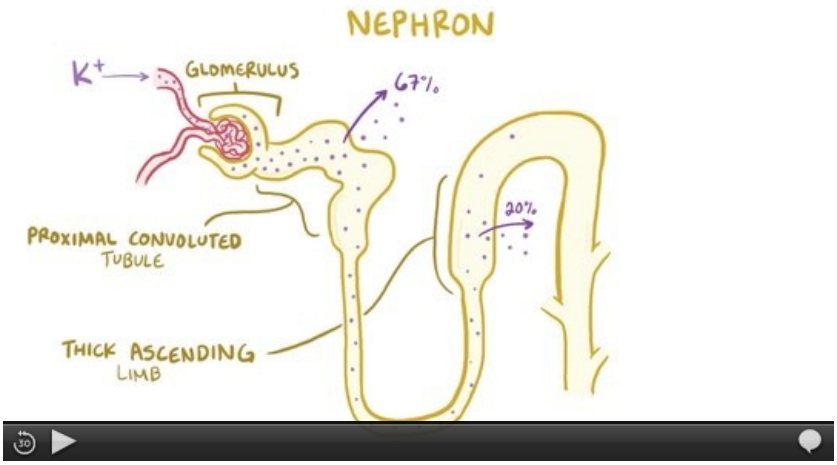
Clinical picture

In general, clinical symptoms depend on the rate of decline in potassium. Acute decline is accompanied by severe symptomatology, chronic deficit is well tolerated.

- **Cardiac symptoms:** conduction and heart rhythm disorders. On the ECG we find low, flattened to inverted T waves, positive U waves, prolongation of the QT segment. The changes are best seen in II. drain. Supraventricular and ventricular extrasystoles occasionally occur, the risk is potassium < 3 mmol/l.
- **Neuromuscular symptoms:** muscle weakness to paralysis, including involvement of the respiratory muscles with the development of respiratory insufficiency. Dysfunction of smooth muscles leads to constipation and even paralytic ileus, with potassium < 2 mmol/l, muscle ischemia with subsequent rhabdomyolysis may occur as a result of insufficient vasodilation response to exercise .
- **Metabolic manifestations:** hypokalemia inhibits the release of insulin , i.e. glucose tolerance decreases. Due to its effect on protein metabolism, chronic hypokalemia is the cause of growth failure.
- **Renal manifestations:** kaliopenic nephropathy, reduced concentration capacity of the kidneys (polyuria , polydipsia, thirst).
- **Endocrine manifestations:** the production of aldosterone and insulin decreases, the production of renin increases .

Potassium changes by about 0.5 to 0.6 mmol/l when the pH changes by about 0.1!

Summary video



Video in English, definition, pathogenesis, symptoms, complications, treatment.

Diagnostic algorithm

In the differential diagnosis of hypokalemia, we use values of potassium waste in urine, HR, ABR values and blood pressure.

K < 3.5 mmol/l + non-constant UK

- RAL
- TIMES
- familial periodic paralysis
- insulin
- β-agonists

Transmembrane movement of potassium from ECT to ICT , i.e. the total amount of potassium is normal.

K < 3.5 mmol/l + UK < 20 mmol/l

- extrarenal losses
    - skin loss (e.g. patients with cystic fibrosis)
    - losses from the GIT (vomiting, diarrhea)
- reduced supply
    - starvation
    - anorexia nervosa

The total amount of potassium is reduced, UK < 10–20 mmol/l indicates maximum saving of potassium by the kidneys. The current finding of MAC is typical for potassium losses during diarrhea, from stomas, fistulas, etc.

K < 3.5 mmol/l + UK > 20 mmol/l = renal losses

- normal BP
    - normal BP + HCO 3 - > 22 mmol/l: Bartter's syndrome , Mg depletion , osmotic diuresis, drug (diuretics, amphotericin B)
    - normal BP + HCO 3 - < 22 mmol/l: renal tubular acidosis
- hypertension
    - hypertension + low plasma renin activity: primary hyperaldosteronism , Liddle's syndrome , Cushing's syndrome
    - hypertension + increased plasma renin activity: renovascular hypertension , renin-secreting tumors, malignant hypertension

The total amount of potassium is reduced, UK > 20 mmol/l, excretion fraction FE-K > 0.3 (the norm is < 0.1) and the ratio U-Na/UK < 1 indicates renal potassium loss. The combination of hypokalemia + MAL + hypertension is a typical finding for primary hyperaldosteronism or hyperreninemia. Accurate determination of the diagnosis requires examination of the renin-angiotensin-aldosterone system . Conversely, the combination of hypokalemia and MAC can indicate RTA, DM ketoacidosis with increased renal potassium loss.

Therapy

We fundamentally try to influence the underlying cause of hypokalemia. We always orient ourselves according to ECG and ABR, we never treat potassium "in isolation". In practice, the total potassium deficit is very poorly estimated, so we perform the correction very carefully. In MAL with transmembrane transfer, ABR adjustment is sufficient to correct potassium.

In mild hypokalemia, we prefer after substitution. We serve potassium preparations, fruit (bananas, oranges, dried fruit), juices, tea.

A true potassium deficiency and the need for its replacement is evidenced by hypokalemia in association with MAC.

When K < 3 mmol/l in patients with clinical manifestations (neuromuscular, cardiac symptomatology), in patients intolerant *to potassium,* we correct the deficit *iv* . To inhibit renal losses of potassium, we use potassium-sparing diuretics (spironolactone, amiloride). These diuretics have only a weak diuretic effect by themselves, so it is advantageous to combine them with loop diuretics (furosemide).

- **formula for calculating potassium deficit:** potassium in mmol = (normal potassium − current potassium) x 0.3 x kg th
- we pay about 1/2 of the calculated amount

Potassium is given in a concentration of max. 40 mmol/l (**ie max. 2 amp. 7.5% KCl per 10 ml in 500 ml of solution**), a higher concentration leads to the development of phlebitis . We do not exceed a speed of 30 mmol/hour, i.e. a maximum of 0.5 mmol/kg/hour. **The usual maximum rate is 20 mmol per hour, and a safe initial bolus is 20 mmol** over 30 minutes in an adult.

Links

Related articles

- Hyperkalemia
- Potassium

Source

- HAVRÁNEK, Jiří: *Dysbalance kalia*. (upraveno)
- RYŠAVÁ, Romana, Interní Med. 2006; 9: 385–388 (https://www.internimedicina.cz/pdfs/int/2006/09/04.pdf)