

Hypokalaemia

Hypokalaemia is defined as $K^+ < 3.5 \text{ mmol/l}$ (lower and upper potassium limits may vary slightly with respect to local laboratory reference limits). It can be a real potassium deficiency or a simple transfer of extracellular potassium into the cells. Potassium values should always be corrected for the ABR values and the ECG curve.

Potassium values should always be related to pH values and ECG shape.

=Etiopathogenesis

- **Transcellular potassium transfer:** acute changes in ABR affect the transcellular potassium distribution. Therefore, both acute MAL and RAL are often accompanied by severe hypokalaemia due to the transfer of potassium into the cell in exchange for hydrogen ion.
- **Insufficient potassium intake:** is a rare cause of hypokalaemia today. It was observed in infants who received commercially produced low-chlorine cow's milk; hypochloraemic MAL and hypokalaemia developed.
- **Kidney hardening losses:** causes administration of diuretics, overactivity mineralocorticoids, RTA, hyperreninemia, diabetic ketoacidosis.
- **Potassium losses from the GIT:** vomiting and / or diarrhea.
- A rare cause of hypokalaemia is '*hypokalaemic periodic paralysis*'. It is a disease with AD heredity. The basis is a mutation of the α -1S subunit of the muscle cell calcium channel T-tube or a mutation of the ryanodine receptor, which controls the release of calcium from the sarcoplasmic reticulum.

Clinical picture

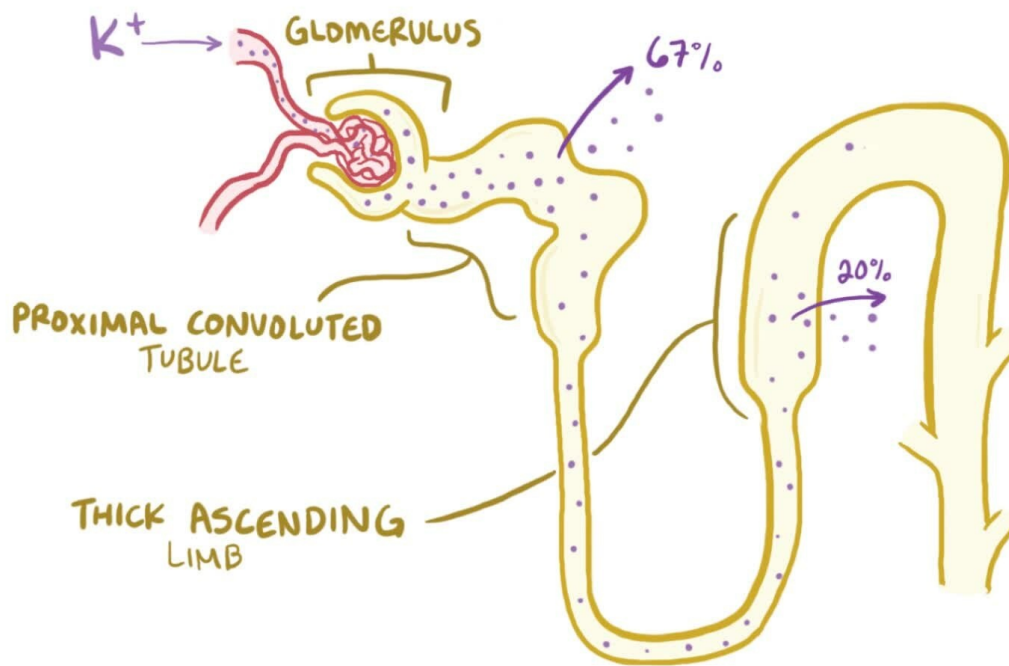
In general, clinical symptoms depend on the rate of decrease in potassium. Acute decline is accompanied by severe symptomatology, chronic deficit tolerates well.

- **Cardiac symptoms:** conduction and heart rhythm disorders. On the ECG we find low, flattened to inverted T waves, positive U waves, QT elongation. The changes are best seen in II. leadership. Supraventricular and ventricular extrasystoles occur occasionally, with a risk of potassium $< 3 \text{ mmol/l}$.
- **Neuromuscular symptoms:** muscle weakness to paralysis, including respiratory muscle involvement with respiratory insufficiency. Smooth muscle dysfunction leads to constipation to paralytic ileu, at a potassium level $< 2 \text{ mmol/l}$, due to insufficient vasodilatory response to exercise, muscle ischemia with subsequent rhabdomyolysis can occur.
- **Metabolic manifestations:** hypokalaemia inhibits the release of insulin, ie glucose tolerance decreases. Due to its effect on protein metabolism, chronic hypokalaemia is the cause of growth failure.
- **Renal manifestations:** calicopenic nephropathy, decreased renal concentration (polyuria, polydipsia, thirst).
- **Endocrine manifestations:** decreases the production of aldosterone and insulin, increases the production of renin.

Potassium changes by about 0.5 to 0.6 mmol/l with a pH change of about 0.1!

Summary video

NEPHRON



Diagnostic algorithm

In the differential diagnosis of hypokalaemia we use the values of urine potassium waste, U-K, ABR values and blood pressure. $K < 3.5 \text{ mmol/l}$ + non-constant U-K

- RAL
- MAL
- familial periodic parylsis
- insulin
- & beta; -agonists

Transmembrane transfer of potassium from ECT to ICT, ie the total amount of potassium is normal.

$K < 3.5 \text{ mmol/l}$ + $U-K < 20 \text{ mmol/l}$

- extrarenal losses
 - skin loss (eg patients with cystic fibrosis)
 - GIT losses (vomiting, diarrhea)
- reduced supply
 - starvation
 - anorexia nervosa

The total amount of potassium is reduced, $U-K < 10-20 \text{ mmol/l}$ indicates the maximum saving of potassium by the kidneys. The current finding of MAC is typical for potassium losses in diarrhea, from stoma, fistula, etc.

$K < 3.5 \text{ mmol/l}$ + $U-K > 20 \text{ mmol/l}$ = renal losses

- normal BP
 - normal BP + $\text{HCO}_3^- > 22 \text{ mmol/l}$: Bartter's syndrome, depletion Mg, osmotic diuresis, pharmaceuticals (diuretics, amphotericin B)
 - normal BP + $\text{HCO}_3^- < 22 \text{ mmol/l}$: 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

Total potassium is reduced, $U-K > 20 \text{ mmol/l}$, excretion fraction $\text{FE-K} > 0.3$ (norm is < 0.1) and $U\text{-Na} / U\text{-K}$ ratio < 1 indicates renal potassium loss. The combination of hypokalaemia + MAL + hypertension is a typical finding for primary hyperaldosteronism or hyperreninemia. Accurate diagnosis requires examination renin-angiotensin-aldosterone system. Conversely, a combination of hypokalaemia and MAC may indicate RTA, DM

ketoacidosis with increased renal potassium loss.

Therapy

We fundamentally try to influence the root cause of hypokalemia. We always orient ourselves according to the ECG and ABR, we never treat potassium "in isolation". In practice, the total potassium deficit is very poorly estimated, so we make the correction very carefully. In MAL with transmembrane shift, an ABR adjustment is sufficient to correct for potassium. Hypokalaemia associated with MAC indicates a real potassium deficiency and the need for its replacement. In mild hypokalaemia, we prefer p.o. substitution. We serve potassium, fruit (bananas, oranges, dried fruits), juices, tea.

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At $K < 3$ mmol/l in patients with clinical manifestations (neuromuscular, cardiac symptoms), in patients intolerant to *after* we correct the deficit *iv* We use potassium-sparing diuretics (spironolactone, amiloride) to inhibit renal potassium loss. These diuretics alone have only a weak diuretic effect, so we preferably combine them with loop diuretics (furosemide).

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

Potassium is administered in a concentration of max. 40 mmol/l (ie max. 2 amp. 7.5 % KCl in 10 ml to 500 ml of solution), higher concentration leads to the development of phlebitis. We do not exceed a rate of 0.5 to 1 mmol/kg/hour

Links

related articles

- Hyperkalaemia
- Potassium

External links

- Template:Acute
- Hypokalémia a EKG (TECHmED) (<https://www.techmed.sk/hypokaliemia/>)

Source

- HAVRÁNEK, Jiří: Dysbalance kalia. (upraveno)