PATHOPHYSIOLOGICAL ASPECTS IN HYPER-AND HYPOKALEMIA

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**Abstract:** Potassium, the most abundant intracellular cation is essential for the life. Potassium is obtained through the diet. Gastrointestinal absorption is complete, resulting sometimes an excess intake of about 1mEq/kg/day (60-100 mEq). This excess is excreted through the kidneys (90%) and the gut (10%). Potassium homeostasis is maintained predominantly through the regulation of renal excretion. The most important site of regulation is the cortical collecting tubule, where aldosteron receptors are present. The importance of potassium in the human body is reflected through the main role in the rest potential, in the ionic and fluid balance and in the pH balance. The potassium disorders can determine severe, sometimes life-threatening clinical situations. We propose to present the pathological mechanisms that appears in the potassium disorders.

**INTRODUCTION**

Potassium is a major intracellular cation, with intracellular levels between 150 to 160 mEq / L. Extracellular, normal serum potassium values are 3.5-5.5 mEq / L, obviously, they do not reflect the total amount of body potassium. Total deposit of K\(^+\) is about 50 mEq / kg. (1).

Adjustment is made by K\(^+\) pump Na \(^+\) / K\(^+\) ATP-aza, controlled by insulin receptors or beta-2 adrenergic receptors.

**Keywords:** kalemia, hyperkalemia, hypokalemia

**Cuvinte cheie:** potasemie, hiperpotasemie, hipopotasemie

**Rezumat:** Potasiul, cationul major intracelular, este esențial pentru viață. Obținut prin aport alimentar, potasiul este absorbit complet din tractul gastrointestinal, realizând uneori situații cu exces de aport, mai mari decât necesarul zilnic de 1mEq/kg/zi (60-100 mEq). Excesul este eliminat apoi pe cale renală (90%) și fecală (10 %). Homeostazia potasiului este menținută în principal prin reglarea excreției renale, la nivelul corticalei tubilor colectori, unde există receptori pentru aldosteron. Importanța potasiului în organism se reflectă prin rolul său principal în echilibrul ionic și lichidian intracelular și în realizarea echilibrului pH-ului. Dezechilibrele potasemiei determina situații clinice cu diferente grade de severitate, uneori amenințătoare de viață. Articolul de față propune explicarea principalelor mecanisme fiziopatogenice întâlnite în dezechilibrele potasemiei.

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**Figure no. 1. EMPA Residency UTHSCSA- Electrolyte Disorders-Fluid Compartments-slide 4**

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**Hyperkalaemia** defined serum K\(^+\) levels> 5.5 mEq / L, is caused by an excess of total K\(^+\). Is explained by 3 mechanisms that are intricated: the increase intake of K\(^+\), the decreased excretion and the shift of K\(^+\) from intracellular to extracellular space. Although the most common cause is a deterioration of renal excretion, hyperkalaemia may occur as a result of metabolic acidosis or uncontrolled diabetes mellitus. (2)

THE EXCESSIVE INTAKE OF POTASSIUM is an unusual mechanism, because the shifting the K\(^+\) intracellular and the renal excretion, allow a person with intact homeostatic mechanisms to ingest virtually large quantities of potassium without changing its potasemia. Even the parental administration of potassium creates minimal increase in serum potassium concentration.

DECREASED EXCRETION OF POTASSIUM: most often associated with increased intake of potassium is the most common cause of hyperkalaemia. Is encountered in acute or chronic kidney failure, after administration of drugs (K\(^+\) sparing diuretics, ACEI, NSAIDs), in Addison’s disease, LES or because of inadequate response to aldosterone in the distal renal tubule (type IV of renal tubular acidosis) in DZ, or chronic partial urinary tract obstruction. (3)

SHIFT OF K\(^+\) FROM INTRACELLULAR TO EXTRACELLULAR SPACE: a rare and isolated mechanism can exacerbate the hyperkalaemia caused by increased ingestion and / or decreased renal excretion. Clinical situations include hyperosmolarity, hyperglycemia, moderate or severe physical exercises, digitalis intoxication, tumor lysis syndrome, acute intravascular hemolysis or rhabdomyolysis.

In case of metabolic acidosis, hyperkalaemia appears more common in acidosis caused by mineral acids (HCl,
appears also in leukocytosis or thrombocytosis. (6) Excretion of K+ in the urine is complex: the gastric juice contains potassium, which is released into the blood after the use of laxatives or intestinal diverticulosis, vomiting and diarrhea. Other causes of renal potassium losses are consecutive to the administration of high doses of penicillin, theophylline lithium or other medications (amphotericin, carbenicillin). Other causes of renal potassium losses are consecutive to the administration of medications (amphotericin, carbenicillin) or the use of laxatives or intestinal diverticulosis, vomiting and diarrhea. Other causes of renal potassium losses are simultaneous with the use of potassium-sparing diuretics and medications (amiloride, triamterene, spironolactone). The Bartter and Gitelman syndromes are genetic syndromes that are characterized by severe hypertension and hypokalemia. It is caused by decreased reabsorption of Na in the distal nephron.

**Hypokalemia**

Hypokalemia causes low intake, excessive loss of K+ through the gastrointestinal tract and kidneys, excessive penetration of K+ in the intracellular space, diluting the blood or administration of β-blockers. All these cases cause neuromuscular irritability, cardiac effects, acid-base imbalance, or effects on renal metabolism. Hypokalemia can be explained by 3 mechanisms: **The low intake of K+** which is rare and occurs in cases of severe cases of increases or decreases of K+, without a frank hypokalemia or hyperkalemia. This occurs due to chronic diuretic therapy (thiazide, loop diuretics) or osmotic diuresis. These drugs most commonly cause hypokalemia. It's blocking Na+ reabsorption in the distal nephron.

Hyperkalemia increases K+ by direct effect of mineralocorticoids on secretion K+ in the distal nephron (Cushing's syndrome, primary aldosteronism or, rarely, renin-secretant tumors). Congenital adrenal hyperplasia, causes hypokalemia by training in excess of mineralocorticoid. Liddle syndrome is an autosomal dominant disease characterized by severe hypertension and hypokalemia. It is caused by increased reabsorption of Na in the distal nephron, with hypertension and renal loss of K+. (8) The Bartter and Gitelman syndromes are genetic disorders characterized by renal loss of K+ and Na+, excessive production of renin and aldosterone, but normal values of arterial pressure. (9) In renal tubular acidosis and Fanconi syndrome, loss of K+ will join those of glucose, phosphate, uric acid and amino acids.

Other causes of renal potassium losses are consecutive to the administration of medications (amiloride, triamterene, spironolactone) or administration of high doses of penicillin, theophylline lithium thalidomide, and dopamine.

Gastrointestinal losses by chronic diarrhea, chronic use of laxatives or intestinal diverticulosis, vomiting and malabsorption are common causes of hypokalemia. In vomiting, the mechanism is complex: the gastric juice contains potassium, approx. 10 mEq / L. The vomiting causes volmic depletion and metabolic alkalosis and is associated with increased renal excretion of K+. Vomilec depletion produce secondary hyperaldosteronism with increased secretion of K+ in the tubular collectors, so the increase of Na+ reabsorption. Metabolic alkalosis increases K+ secretion in the tubular collectors, by decreasing the availability of hydrogen ions in response to Na+. reabsorption. Gastrointestinal losses may be simultaneous with the kidney by metabolic alkalosis and stimulation of aldosterone due to depletion volenice. (10)

Trauma and excessive sweating also causes increased loss of K+.

**SHIFT OF K+ FROM EXTRACELLULAR TO INTRACELLULAR SPACE** is frequently associated with increased excretion of K occurs in metabolic alkalosis or if increasing the dosage of insulin in diabetes mellitus. Intracellular penetration of potassium is often episodic and self-limited (eg insulin therapy in acute hyperglycaemia). On the pathophysiological, to increase the pH in the extracellular space, potassium enters the cell. Increase with 0.10 in pH, cause decreases by 0.5 mEq / L of serum potassium.

Stimulation of the sympathetic nervous system, particularly by β2-agonists cause hypokalemia. (eg, albuterol, Terbutaline) due to increased cellular potassium acquisition. Similarly, in patients with thyrotoxicosis, hypokalemia appears excessive stimulation β-sympathomimetics (thyrotoxicosis, hypokalemia periodic paralysis). Periodic familiar paralysis is a rare disease of autosomal dominant, characterized by transient episodes of severe hypokalemia due to high intracellular penetration of K+. In these episodes occur at different stages of paralysis. Are precipitated by high carbohydrate diet and intense exercise, but may occur without precipitating factors. (11) Since potassium is predominantly an intracellular cation, regulated by different mechanisms, may encounter severe cases of increases or decreases of K+, without a frank hypo / or hyperkalemia. Arguably, the serum potassium imbalnces do not always reflect real excess or deficit of total deposit of K+ in the body.

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