Calcium (General)

Calcium is one of the most important extracellular ions. Intracellularly, the concentration of calcium is several orders of magnitude lower than outside the cell, but a temporary increase in the cytoplasmic concentration plays a vital role in cell signaling (calcium serves as a second messenger mediating the effect of hormones , cytokines and other mediators) or, for example, in the control of muscle contraction. Calcium creates the structure of bones and teeth , participates in the regulation of neuromuscular activity, coagulation, heart activity. [1] In the body, calcium makes up about 1.5% of the total body weight, and more than 99% of it is in the bones. [2]Calcium is contained in milk, cheese, eggs and "hard water". [3]In the intestine, the necessary amount of calcium is absorbed from food, and the rest is excreted in stool and urine.

Reference limits:

- serum: **2.25–2.75 mmol/l** (ionized **1–1.4** mmol/l)
- urine: **2.5–7.5 mmol/day.**

3 hormones are involved in maintaining serum calcium levels: parathyroid hormone , vitamin D and calcitonin . These hormones regulate calcium absorption in the intestine, reabsorption in the kidneys, excretion and utilization of calcium in the bones. [4]

Importance of calcium

The importance of calcium and physiological functions linked to the presence of calcium: inorganic component of bones and teeth, blood clotting factor (factor IV), formation of kinins, regulation of enzymes, release of hormones and their effect, regulation of excitation of a number of tissues, regulation of transmitter release, activates troponin in skeletal muscle , the formation of tropomyosin , which activates actin, activates contraction in smooth muscle by binding to calmodulin. Intracellularly, calcium participates in the action potential of the cell, in contraction, in motility, in cell division, in the structural integrity of the cell, and in the increase of glycolysis. A decrease in the extracellular concentration of calcium increases neuromuscular excitability and thus also the possibility of tetany .[2]

Calcium resorption

The recommended daily dose for adults is around 1 g. Its absorption is physiologically around 25-40%. Calcium is resorbed actively in the duodenum and jejunum, passively in the ileum and colon. Calcium resorption takes place simultaneously with its secretion. Alkaline pH significantly reduces calcium resorption.[2]

At the enterocyte level, calcium resorption occurs in two ways:

1. **Transcellular** - On the side facing the intestinal lumen, the specific transport protein calbindin is used. On the side of the basolateral membrane, calcium is then actively transported in the presence of energy against the drop in concentration in the blood.
2. **Paracellularly** – Calcium is transported directly from the intestinal lumen, and calcium is transported which reaches the paracellular space by being released from the lysosome of the enterocyte. [2]

Calcemia

Calcium is in the blood in three forms:

1. 50% in ionized form (most biologically active, able to diffuse through biological membranes, decisive for neuromuscular irritation);
2. 40% bound to proteins (not freely diffusible);
3. 10-13% in the form of complexes (as bicarbonate, phosphate, citrate).

Blood alkalosis results in increased binding of calcium to plasma proteins, thereby reducing free, ionized calcium, but the total calcium concentration is unchanged. [2]

The binding of calcium to proteins depends on the pH of the blood – when the pH rises (alkalosis), more binding sites for Ca 2+ are released on the proteins , and therefore ionized Ca 2+ decreases . Therefore, for example, as a result of hyperventilation, tetany occurs[3] . Hypoalbuminemia is associated with a decrease in calcium, but there are no symptoms of hypocalcemia because the ionized form is normal. [5]

Phosphate metabolism is closely related to calcium metabolism. Intravenous administration of phosphates lowers the serum Ca 2+ concentration because calcium phosphate is formed, which is deposited in the bones. Conversely, hypophosphatemia causes hypercalcemia by releasing Ca 2+ from the bones. [3]

Calcium excretion

Only ionized calcium can be filtered by the kidneys . In the region of the proximal canal, reabsorption takes place both transcellularly (15–20%, actively) and paracellularly (80–85%, passively). The ascending part of Henle's loop reabsorbs calcium transcellularly and paracellularly.

Both parathyroid hormone and calcitonin stimulate reabsorption via a transcellular pathway in this area of the nephron . In the distal part of the nephron, parathyroid hormone , calcitonin and calcitriol are involved in reabsorption of calcium . [2]

Maintaining calcium homeostasis

Calcium homeostasis is maintained by the activity of osteoclasts , which resorb bone and thus increase the concentration of calcium in the serum, and by the simultaneous activity of osteoblasts , which participate in the new formation of bone mass, thereby reducing the concentration of calcium in the serum. Hormones that maintain calcium homeostasis include calcitonin, parathormone and vitamin D with the participation of the intestines, kidneys and bones. Regular and adequate physical activity of the organism is also important for maintaining calcium homeostasis , which improves calcium resorption from the intestine by increasing blood flow to the splanchnic area and the formation and maintenance of bone mass quality. Calcium entry into cells can be reduced pharmacologically by calcium channel blockers . Magnesium is an antagonist of calcium. [2]

Disorders of calcium homeostasis

Hypocalcemia

See the Hypocalcemia page for more detailed information .

Hypocalcemia is a serum calcium level **lower than 2.14** mmol/l.

Causes :

- hypoalbuminemia (nephrotic syndrome , ...), hypoparathyroidism , pseudohypoparathyroidism, hypomagnesemia , vitamin D deficiency , serum calcium depletion (osteoblastic metastases, pancreatitis , ...);
- alkalosis , after transfusions (citrate balances Ca 2+).

Clinical signs:

- neuromuscular and psychiatric symptoms: paresthesia , tetany , positive Chvostk sign , shortness of breath , confusion , irritability;
- rickets , osteomalacia , increased tooth decay, hypoplastic dentition;
- when evaluating, it is always necessary to look at: pH , Ca 2+ in serum, ionized fraction of Ca 2+ and total protein;
- ECG: **prolongation of the QT interval** .

Therapy:

- infusion of 10% *Calcium gluconicum* , or 10% *Calcium chloratum* CaCl 2 · 6 H 2 O 10% every 12 or 6 hours;
- heart action monitoring –**risk of bradycardia** with rapid administration .

Hypercalcemia

More detailed information can be found on the Hypercalcemia page .

Causes:

- hyperparathyroidism , hypervitaminosis D , osteolytic metastases, hyperproteinemia

Clinical signs :

- GIT symptoms;
- CNS symptoms: weakness , lethargy , fatigue, confusion, reduction of neuromuscular excitability, vomiting
- ECG: **shortening of the QT interval**
- calcemia above 4 mmol/l – **hypercalcemic crisis**: confusion, unconsciousness , cardiac arrest in systole (so-called " **chemical death** ").

Therapy :

- **hyperhydration** (physiological solution), diuretics (furosemide), **alkalization** , dialysis.

Links

Related Articles

- Disorders of calcium phosphate metabolism

- Bone and calcium metabolism
- SCHNEIDERKA, Petr, et al. *Chapters in clinical biochemistry.* 2nd edition. Prague: Karolinum, 2004. ISBN 80-246-0678-X .

Other Literature Used

- SCHNEIDERKA, Petr, et al. *Chapters in clinical biochemistry.* 2nd edition. Prague: Karolinum, 2004. ISBN 80-246-0678-X .

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