Hypocalcemia

The reference values for calcium are usually **2.25-2.75 mmol/l**. Of this, about 40-45 % of calcium is bound to proteins in the serum, 5-10 % is in complexes with acid anions and 50 % is in ionized form. The ionized fraction of serum calcium is crucial because only this calcium can affect various ionic processes, such as normal muscle contractility including myocardium and stability membrane charge. When ionized calcium **drops** below 1 mmol/L, ['neuromuscular excitability]] **increases**, but also myocardial contractility. In skeletal muscle, there is a change in charge sarcoplasmic reticulum and the release of Ca^{2+} (leading to contraction). In myocardium, on the other hand, there is a lack of Ca^{2+}ions (intense exchange with extracellular space). The waste urine is about 5 mmol/24 hours. The key role in the interpretation of effective calcium is the pH of the blood, which is crucial for the appropriate proportion of the ionized calcium fraction. We refer to Ca <2.14 mmol/l as **hypocalcemia**.

Hypocalcemia:

Etiology

Hypoalbuminemia

Kidney and Liver diseases lead to insufficient synthesis and loss of Plasma proteins (especially albumin). This reduces the overall binding capacity of serum.

Causes

- nephrotic syndrome
- exudative enteropathy
- liver cirrhosis

🔎 *For more information see Hypoalbuminemia.*

Endocrine origin

Hypoparathyroidism

- **idiopathic** - sporadic and familial
- **infiltration** - hemochromatosis, amyloidosis, Wilson's disease, metastases
- **iatrogenic** - surgical removal of all four parathyroid glands (thyroidectomy error)
- **autoimmune** - autoimmune polyglandular syndrome I
- **anomalies** - aplasia, DiGeorg's syndrome

Pseudohypoparathyroidism is a condition in which target cells do not respond to parathyroid hormone.

Vitamin D deficiency

- nutritional deficiency
- GIT disorders
- chronic renal failure
- vitamin D-resistant rickets
- vitamin D-dependent rickets
- nephrotic syndrome

- acceleration of vitamin D metabolism - anticonvulsants, alcohol, cholestyramine

Serum calcium depletion

- hyperphosphatemia: dietary excess phosphorus (dairy products), cell lysis syndrome, osteoblastic metastases
- "hungry" bone syndrome
- pancreatitis - soap formation with fatty acids

Critically ill patients

- Gram negative sepsis
- toxic shock syndrome

Other origins

- hypomagnesemia
- Kenny-Caffey syndrome

Pathogenesis

Hypoalbuminemia is associated with a decrease in serum total calcium concentrations, but the concentration of ionized calcium remains unchanged. Therefore, the patient has no symptoms of hypocalcaemia. In alkalosis **, calcium binding to** and especially to the endogenous anion complex increases at the expense of the ionized fraction. Therefore, the patient will have clinical signs of hypocalcaemia even with unchanged total Ca.

Hypomagnesemia reduces parathyroid hormone secretion (PTH is used) and inhibits the bone response to PTH, thus leading to hypocalcemia. Hyperphosphatemia leads to the transfer of calcium phosphate into the tissues, thus also leading to hypocalcemia.

In pancreatitis, calcium is taken up in **organ calcifications** (Balser necrosis). Hypocalcaemia in sepsis is multifactorial:

1. acquired parathyroid insufficiency
2. function of 1-α-hydroxylation in the kidneys
3. loss of sensitivity to vitamin D, etc.

Hypocalcemia in sepsis is considered a poor prognostic feature.

Clinical picture

Symptoms of hypocalcaemia can be **varied**, sometimes noticeable. Chronic hypocalcemia may escape correct diagnosis for several years. Most common symptoms:

- neuromuscular and psychiatric symptoms
- Tetania = central and peripheral nervous system irritability syndrome (generalized tonic spasms, carpopedal spasms, obstetric hand, laryngospasm, dysarthria)

It may be a disorder of intelligence, myopathy. With long-term hypocalcaemia, extrapyramidal symptoms (parkinsonism, choreoatetosis) and bone changes (rachitis, osteomalacia) may occur. There are also changes in the teeth, especially **extreme caries** and **hypoplastic teeth** . In dermatological symptomatology we observe dermatitis, eczema, **psoriatic symptoms**, **brittle hair**, **alopecia areata**. Ocular changes include cataract, **neuritis opti**, **papillary edema** . Cardiovascular symptoms include hypotension, decreased contractility, prolonged QT interval on the ECG. Clinical manifestations are significantly exacerbated (sometimes imitated) by current hypomagnesemia.

Diagnostics

- serum calcium, including ionized fraction
- phosphorus, magnesium, albumin
- creatinine, creatinine clearance
- parathyroid hormone
- parathyroid ultrasound
- vitamin D metabolites
- increase in cAMP in urine after PTH infusion (diagnosis of pseudohypoparathyroidism)

Diagnostic algorithm

1. we examine serum albumin, the level of albumin is necessary to demonstrate hypoalbuminemia, we continue at a normal albumin value of 35–55 g/l
2. we determine serum magnesium, the level of magnesium is necessary to prove hypomagnesemia, at the normal value of magnesium we continue
3. determine serum phosphorus and PTH

- increased PTH + hypophosphataemia → vitamin D deficiency
- increased PTH + normo- or hyperphosphatemia → pseudohypoparathyroidism
- decreased PTH + hyperphosphatemia → hypoparathyroidism

Therapy of acute hypocalcemia

Acute hypocalcemia is a condition requiring **immediate intensive care** for high risk of seizures and laryngospasm.

We solve acute hypocalcemia by administering **10% Calcium gluconicum** (1 ml = approx. 0.2 mmol) 1 ml/kg **or 10% Calcium chloratum** (1 ml = approx. 0.5 mmol) 0 , 2 ml/kg i.v. within 10 minutes (maximum single dose is 10 mmol, maximum rate 1 ml/min.). At the same time, we monitor the heart rate (there is a risk of bradycardia with rapid administration). The dose can be repeated after ECG monitoring after 60 minutes. We usually prefer Calcium gluconicum, because Calcium chloratum is highly irritating to blood vessels and there is a greater risk of tissue necrosis in case of paravenous leakage.

10% MgSO4 0.2-0.5 ml/kg i.v. within 15-20 minutes we administer **with confirmed hypomagnesemia**, ie Mg <0.5 mmol/l or if i.v. administration of calcium does not lead to a remission of clinical symptoms.

After acute treatment, we continue with substitution in drip infusions. If hyperphosphatemia is present, we try to reduce it with a low-protein diet, calcium carbonicum. Hyperphosphatemia has an antagonistic effect on the treatment efforts of hypocalcemia and, in addition, there is a risk of calcium phosphate precipitation in the parenchymal organs.

Hyperventilation tetany

The first therapeutic measure is rebreathing CO_2 by means of a plastic bag, due to the anxiety diazepam *0.1-0.2 mg/kg is also suitable (mostly for adolescents 5-10 mg* pro dosi*) i.v., e.v. additionally calcium.*

Links

Related articles

- Hypercalcemia
- Disorders of calcium-phosphate metabolism
- Pathophysiology of bone, calcium and phosphates
- Trousseau's sign

External links

- Hypokalcemia a EKG (TECHmED) (https://www.techmed.sk/hypokalcemia/)

Source

- HAVRÁNEK, J.: Dysbalance ostatních iontů.