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HYPOPARATHYROIDISM

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Abstract: Hypoparathyroidism is an endocrine disease characterized by a decrease in the production of parathyroid hormone by the parathyroid glands or tissue resistance to its effects, which is accompanied by a violation of phosphorus-calcium metabolism, the main etiology of hypoparathyroidism is damage to the parathyroid glands or is thrown away. neck organs. Since thyroid cancer, primary hyperparathyroidism and other pathologies of the neck organs are common, their radical treatment can lead to the development of hypoparathyroidism, the number of patients with this pathology is expected to increase and increase steadily. Autoimmune hypoparathyroidism is the second most common form of the disease and usually occurs as part of autoimmune polyglandular syndrome type 1.

Key words: thyroid gland, hypoparathyroidism, calcium, vitamin D, treatment.

Hypoparathyroidism is a condition characterized by a decrease in the production of parathyroid hormone (PTH) by the parathyroid glands (PTG) or tissue resistance to its effects, which is accompanied by disturbances in phosphorus-calcium metabolism.

In hypoparathyroidism, the absence or deficiency of PTH is inevitably accompanied by the development of hypocalcemia. The main pathogenetic mechanisms include: a decrease in osteoclast activity with a decrease in calcium release from bones; increased calcium in the urine; suppressing the synthesis of calcitriol in the kidneys and reducing the absorption of calcium from the intestine. PTH deficiency leads to hyperphosphatemia directly due to increased phosphate reabsorption in the renal tubules and indirectly due to hypocalcemia. Chronic hyperphosphatemia in patients with hypoparathyroidism has been shown to be associated with increased circulating levels of fibroblast growth factor 23.

Surgery to the neck organs is the most common cause of hypoparathyroidism, accounting for about 75% of all cases of this disease. Postoperative hypoparathyroidism can result from both direct removal and intraoperative trauma or disruption of the parathyroid gland's blood supply. The risk of chronic hypoparathyroidism is closely related to the number of functional parathyroid glands that remain at the time of surgery: 16% when 1-2 glands are preserved, 6% with 3 glands, and 2.5% with 4 glands.

Autoimmune hypoparathyroidism is the second most common form of hypoparathyroidism caused by immune-mediated destruction of parathyroid cells. It can be a separate disease, but is more common in hereditary autoimmune polyglandular syndrome (APS) type 1. APS type 1 is a monogenic autosomal recessive disease based on a disruption of the autoimmune regulatory gene (AIRE) structure. The pathogenesis of the disease is based on the autoimmune destruction of various endocrine glands, including the parathyroid gland. Type 1 APS is characterized by the classic triad: mucocutaneous candidiasis, hypoparathyroidism, chronic adrenal insufficiency. The disease usually begins in childhood.

Other rare hereditary forms of hypoparathyroidism occur both separately and as part of multicomponent genetic diseases (DiGeorge syndrome, Barakat syndrome, Kenny-Cuffey syndrome, etc.).

Functional hypoparathyroidism, a reversible form of the disease, develops with restoration of PTG function after correction of hypo-/hypermagnesemia in case of magnesium metabolism disturbance.

Rarely, hypoparathyroidism can be caused by infiltrative diseases such as sarcoidosis, amyloidosis, Riedel's thyroiditis, and metastatic lesions of the parathyroid gland. PTG tissue is not sensitive to radiation damage, but very few cases of radiation-related hypoparathyroidism have been described in the literature. Mineral deposits in the parathyroid tissue, such as copper in Wilson's disease and iron in hemochromatosis, are rare causes of hypoparathyroidism. Cases of development of hypoparathyroidism as a result of repeated blood transfusions have been described in patients with thalassemia.

Hypoparathyroidism is a rare disease with an incidence of 0.25 per 1000 population. Available data on the prevalence of hypoparathyroidism are based on large epidemiological studies conducted in the United States, Denmark, Norway, and Italy. The obtained results are relatively similar and show a prevalence of hypoparathyroidism in the range of 23-37 per 100,000 population.

Postoperative hypoparathyroidism is common among women, which is a frequent pathology of the thyroid gland and is associated with thyroidectomy as a result. The prevalence of hereditary forms of hypoparathyroidism does not differ between men and women. No large epidemiological studies have been conducted to assess the prevalence of hypoparathyroidism in the Russian population.

The main clinical manifestations of hypoparathyroidism are caused by the presence of hypocalcemia. An increase in the sensitivity of the sensory neuron is manifested in the form of paresthesia in the limbs and perioral area; motor (motor) neuron - muscle spasms, up to tetany; from classic carpopedal spasm to life-threatening laryngospasm. Severe hypocalcemia is associated with both local and generalized tonic-clonic seizures.

The severity of symptoms depends on the level of calcium in the blood serum, as well as the rate of development of hypocalcemia. The chronic course of the disease is characterized by adaptation to a low level of calcium in the blood serum with the absence of a clear clinical picture even with severe hypocalcemia. In such cases, the factors causing the deterioration of the condition may be increased physical activity, medical procedures, pregnancy and lactation.

The most common complaint of patients with hypoparathyroidism is the presence of cramps and / or paresthesias in the muscles of the upper and lower extremities and in the perioral area. Patients with long-term hypoparathyroidism complain of "brain fog", decreased memory and concentration.

The clinical manifestations of chronic hypoparathyroidism are diverse and affect almost all body systems. Clinical symptoms of chronic hypoparathyroidism may be associated with episodes of hypo- and hypercalcemia, hyperphosphatemia.

The following factors indicate that the patient may have hypocalcemia and hypoparathyroidism:

- surgical intervention in the neck area;
- the presence of paresthesia in the face, upper and lower extremities;
- fibrillary twitching of individual muscles, presence of convulsions in proximal muscles;
- determination of brain calcification;
- for heart rhythm disorders.

Trousseau's positive sign is the appearance of cramping in the arm ("midwife's hand") 1-3 minutes after cuffing the shoulder when measuring blood pressure. This symptom, which is a very sensitive and specific sign of hypocalcemia, is detected in 94% of patients with hypocalcemia and in 1% of patients with normocalcemia. Chvostek's sign - contraction of the facial muscles when hitting the exit of the facial nerve - is a less sensitive and specific sign. A negative Chvostek sign is observed in 30% of patients with hypocalcemia, and a positive sign in 10% of people without pathology.

The diagnosis of hypoparathyroidism is based on the results of laboratory tests!

Criteria for diagnosing hypoparathyroidism.

The most common test for diagnosing hypocalcemia is measuring the total calcium level. In order to exclude false-negative or false-positive results of calcium when the concentration of plasma proteins changes, it is necessary to adjust calcium to the level of albumin in the blood.

If the disease is compensated, it is recommended to carry out dynamic monitoring of total calcium, albumin (with calculation of calcium corrected by albumin), phosphorus, magnesium, creatinine with calculation of GFR every 3-6 months. In the absence of compensation for hypoparathyroidism and / or dose adjustment of standard therapy, it is recommended to evaluate the parameters of phosphorus-calcium metabolism several times a week to assess the adequacy of the selected therapy.

Dynamic monitoring of daily calcium release is recommended every 6-12 months. If hypercalciuria is detected and / or thiazides are prescribed, it is recommended to monitor the amount of calcium in daily urine after 1.5-2 months to assess the adequacy of treatment.

In patients with chronic hypoparathyroidism, treatment with vitamin D and its derivatives and calcium preparations significantly increases the risk of developing nephrolithiasis/nephrocalcinosis.

Patients with a long history of hypoparathyroidism (more than 3-5 years) are recommended to undergo periodic examination by an ophthalmologist to timely diagnose the development of cataracts and determine the need for specialized treatment.

The clinical presentation of calcification of various parts of the central nervous system in patients with long-term hypoparathyroidism is not specific. The most common movement disorders include: muscle stiffness, parkinsonism, hyperkinesis (chorea, tremor, dystonia, athetosis, orofacial dyskinesia); cognitive disorders; cerebellar symptoms and speech disorders. In some cases, epileptic seizures and dementia are observed. A combination of different clinical signs is often observed. The question of the existence of a pathogenetic relationship between neurological symptoms and the size and localization of calcification remains controversial. If these symptoms appear and / or calcification of the basal ganglia is detected in the CT scan of the brain, a consultation with a neurologist is indicated.

Bone turnover is slow in hypoparathyroidism, so in the absence of associated risk factors, such as glucocorticoid therapy, there is no prerequisite for a decrease in BMD over time. In patients with chronic hypoparathyroidism, it is recommended to conduct a comprehensive examination, including the determination of signs of bone turnover and bone radiography, to assess the state of bone tissue.

The main goals of long-term therapy for hypoparathyroidism

Albumin-corrected blood calcium level	In patients without clinical signs of hypocalcemia, keep at the lower limit of the reference range (2.11-2.65 mmol / L) or slightly below.	2.1-2.3 mmol/l
Daily calcium excretion rate (study of calcium levels in daily urine)	Keep within target range	Men: <7.5 mmol/day Women: <6.25 mmol/day
Serum phosphorus levels	Keep in reference range	0.8−1.4 mmol/l
Serum magnesium level	Within the reference range	0.7−1.05 mmol/l
25 (OH) vitamin D level	As in the general population	30–60 ng/ml (75–150 nmol/l)
General well-being and quality of life	Personalized treatment	

- Patients with hypoparathyroidism, as well as the general population, are often characterized by vitamin D deficiency. Therefore, it is recommended to use local forms of vitamin D (cholecalciferol**) to correct it.
- The optimal levels of 25 (OH) D for the population of Russia are set in the range of 30-60 ng / ml (75-150 nmol / l). A 25(OH)D level above 100 ng/mL (250 nmol/L) can cause vitamin D toxicity in the body and is not recommended.
- Treatment with Alfacalcidol**, calcitriol** does not significantly affect the level of 25(OH)D in blood serum.

Standard therapy for hypoparathyroidism includes vitamin D preparations and its derivatives (alfacalcidol**, calcitriol**) and calcium preparations. The calcemic effect of calcitriol** is approximately twice that of alfacalcidol**.

- In order to maintain the amount of calcium in the blood at the target level, it is recommended to titrate the doses of vitamin D drugs and its derivatives, to divide the total dose of the drug into 2-3 doses. Dose titration is usually in increments of 0.5 (or 0.25) mcg for alfacalcidol and 0.25 mcg for calcitriol**. Larger dose adjustments may be required for severe hypo/hypercalcemia. The recommended time interval for adjusting the dose of vitamin D and its derivatives (alfacalcidol**, calcitriol**) is 2-3 days, which is related to adequate assessment of their pharmacokinetics and the resulting changes. In the case of an asymptomatic course and moderate fluctuations in calcium levels, the laboratory can evaluate the adequacy of adjusted doses after 7-10 days. It may take about 2-3 months to evaluate the clinical effectiveness of the selected therapy and to achieve stable calcium values, especially for patients with a high need for vitamin D and its derivatives.
- The use of calcium salts in isolation is not pathogenetically justified and leads to a short-term increase in the amount of calcium in the blood serum.
- Patients with hypoparathyroidism are advised to use a diet rich in calcium-containing foods.
- Various calcium preparations are used to treat hypoparathyroidism. As a rule, preparations containing calcium carbonate (40% elemental calcium), including as part of the combined preparation "calcium carbonate + cholecalciferol" are used in an average daily dose of 1-3 g (higher doses can be used). Dietary supplements of calcium citrate (21% elemental calcium) can also be used. When prescribing, the pharmacokinetics of various drugs should be taken into account: calcium carbonate is better absorbed in the acidic environment of the stomach, so it is preferable to take it with food; Calcium citrate is recommended for patients with achlorhydria or those receiving treatment with proton pump inhibitors.

- High-dose calcium supplementation reduces the need for vitamin D and its derivatives and improves control of maintaining target serum phosphorus levels by sequestering phosphorus in the intestine.

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