Hypoparathyroidism

Hypoparathyroidism is a syndrome caused by insufficient secretion of PTH , which leads to hypocalcemia. It is a relatively rare disease, the incidence of which is 7 cases per 1,000,000 inhabitants per year (autoimmune etiology).

Pseudohypoparathyroidism:

Etiology

Surgical removal of the parathyroid glands is the most common cause, usually a complication of thyroidectomy. Transistor hypoparathyroidism can occur even after removal of the adenoma, when the remaining bodies are in functional depression.

Idiopathic hypoparathyroidism is an autoimmune disorder of the parathyroid glands that can occur alone or as part of autoimmune polyglandular syndrome type 1 .

Familial hypoparathyroidism is an autosomal recessive inherited disorder resulting from a mutation in the PTH gene or a mutation in one of the transcription factors involved in the development of the parathyroid glands. The autosomal dominant disorder is caused by a mutation in the calcium sensing receptor gene. Other less common causes include DiGeorge syndrome , HDR syndrome , thalassemia , severe hypomagnesemia, metastases infiltrating the parathyroid glands, Wilson's disease (copper deposition), and hemochromatosis (iron deposition).

Clinical Picture

Rapidly occurring hypoparathyroidism manifests itself as **tetanic convulsions , increased neuromuscular irritability , carpopedal spasms** , paresthesia of the limbs and face. In the case **of slow development , psychological symptoms** (in chronic forms), **weakness , fatigue , apathy , anxiety , personality** disorders **, vision disorders** (due to cataracts), extrapyramidal disorders are present .

Objectively , we find **Chvostk's** sign **and Trousseau's** sign , hyperactivity of tendon reflexes, dry skin with desquamation, brittle nails, loss of hair and eyebrows, disorders of tooth development in children, cataracts, increased intracranial pressure.

We will demonstrate **hypocalcemia** , **hyperphosphatemia** , **low** serum concentration of **PTH in the *laboratory*** .

Further examination:

- Skiagram of the skull - calcification of the basal ganglia (in chronic forms).
- Eye examination – posterior lenticular cataract, papilledema (in case of intracranial hypertension).
- ECG - **prolonged QT interval** .

In terms of differential diagnosis, we must differentiate between another cause of hypocalcemia and pseudoparathyroidism.

Therapy and prognosis

Acute forms

The principle is a rapid correction of **calcium levels i.v.** (in the form of infusions with *calcium gluconicum 10% amp.*), administration of **calcitriol** , possibly in case of hypomagnesemia, we also add Mg. We monitor the EKG during the treatment.

The prognosis for a disorder treated correctly and in time is good.

Chronic forms

We administer **vitamin D 2** (8,000–150,000 IU/day), **D3**(10,000–50,000 IU/day) or **calcitriol** (0.25–4 µg/day), while simultaneously supplementing with calcium (1–2 g/day), preferably in two daily doses. We correct the treatment based on the monitoring of serum calcium and phosphate concentrations.

Dental disorders, cataracts, basal ganglia calcification and intellectual disorders can no longer be affected.

Links

Related Articles

- Parathyroid Hormone
- Calcium-Phosphate Metabolism

Resources

- ČEŠKA, Richard, Tomáš ŠTULC and Vladimír TESAŘ, and Milan LUKÁŠ, et al.. *Interna.* 3rd edition. Prague: Stanislav Juhaňák - Triton, 2020. ISBN 978-80-7553-780-5 .

Endocrine system