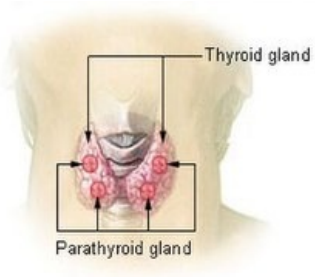
Hyperparathyroidism

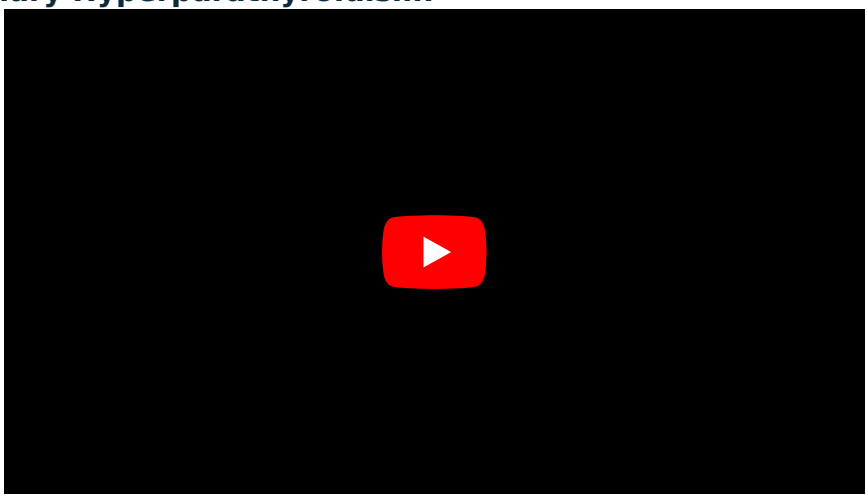
This article has been translated from WikiSkripta; ready for the **editor's review**.

Parathyroid glands are endocrine-active organs whose product is the **parathormone**. Its function is to **regulate calcium metabolism** - it has hypercalcemic effects. It acts on bones, where it **increases osteoclastic resorption**, **increases the absorption of calcium in the kidneys and reduces the absorption of phosphate*s***. In the intestines it **increases the absorption of vitamin D and in the kidneys its hydroxylation** to an active product.

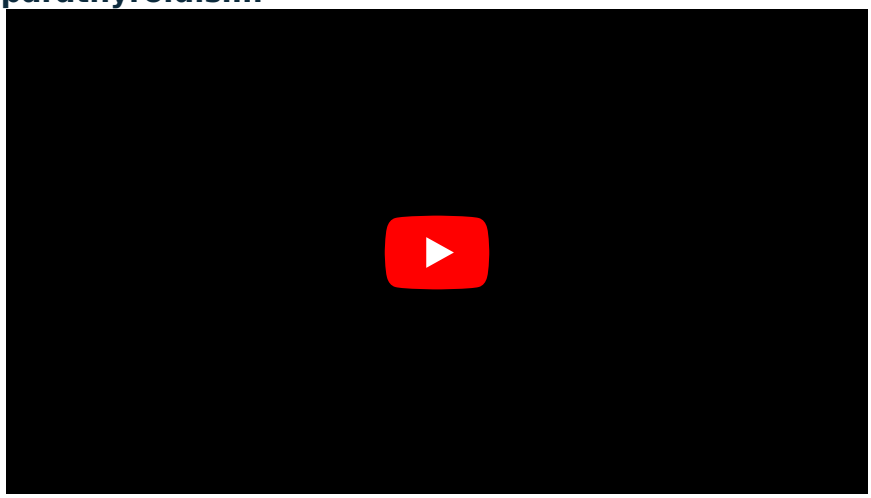


Thyroid and parathyroid glands

Primary Hyperparathyroidism:



Secondary Hyperparathyroidism:



Tertiary Hyperparathyroidism:

Diseases of the parathyroid glands are divided into hyperfunction (**hyperparathyroidism**) and hypofunction (**hypoparathyroidism**).

Primary hyperparathyroidism

Primary hyperparathyroidism arises as a result of **excessive production of parathyroid hormone** and its excessive action on peripheral tissues.

Etiology

The most common cause is an adenoma of one of the corpuscles (80%). It is less often a case of **primary hyperplasia** (15%) and rarely of a **carcinoma** (1-2%). Related to adenoma, it is also necessary to consider the **multiple endocrine neoplasia type I syndrome** (MEN-I; primary hyperparathyroidism, tumors of the endocrine pancreas and tumor of the pituitary gland).

Clinical features

The disease is often diagnosed accidentally during a laboratory examination. If symptoms do develop, they are mainly manifested by **affecting the bones, kidneys and gastrointestinal tract**.

Bone manifestation: is manifested by **bone pain** and **pathological fractures**. Gradual osteoresorption causes the development of **osteoporosis** and a specific bone disorder - **fibrous osteodystrophy** (previously incorrectly referred to as *osteitis fibrosa cystica*). This is characterized by deposits of subperiosteal bone resorption with a typical "pepper and salt" X-ray picture, with severe involvement even brown tumors (osteoclastomas) arise.

Kidney disorders: typical is **nephrolithiasis** (calcium-oxalate stones), less often **nephrocalcinosis** (calcification in the interstitium kidneys). Nephrocalcinosis can lead to renal failure.

Gastrointestinal manifestation: includes more frequent occurrence of **acute pancreatitis** and **peptic ulcer**, which have a complicated course. Dyspepsia, nausea, vomiting or constipation may occur.

Cardiovascular symptoms: hypercalcemia causes **heart rhythm disorders** to **cardiac arrest in systole.** Arterial hypertension occurs more often.

Psychic and neuromuscular symptoms: weakness, fatigue, personality disorders, depression, muscle weakness.

Diagnostics

The basis of the diagnosis is a laboratory examination, which demonstrates **hypercalcemia** (the norm is **2.2-2.7 mmol/l**), **hypophosphatemia** (the norm is **0 .65–1.65 mmol/l**) and an **increase** in the level of serum **PTH**. **Hyperphosphaturia** and **hypercalciuria** are present.

Densitometry and skiagram of the skull and phalanges will clarify bone involvement. USG is sufficient to examine the kidneys.

To detect and localize a tumor or hyperplastic body, we start with a **neck USG** supplemented with a **scintigraphic examination** with **^{99m}Tc-MIBI** *or PET/CT with ^{11}C or ^{18}F* by choline.

Differential diagnosis

We must distinguish individual etiological causes and primary from secondary and tertiary hyperparathyroidism. Furthermore, we exclude other causes of hypercalcemia: *familial benign hypocalciuric hypercalcemia, paraneoplastic production of PTH related peptide/1,25-OH-D/PTH in neoplasia, osteolytic involvement*, and others.

Therapy

The treatment of first choice is **surgical removal of the affected parathyroid gland**. If its localization is known, it is possible to choose a minimally invasive approach, otherwise bilateral exploration of the neck is chosen. After removal of the adenoma, the calcium level can be expected to drop to the physiological range within 24-48 hours. If the patient suffered from significant demineralization of the skeleton before the operation, **severe hypocalcemia**, **the so-called hungry bone syndrome**, **may develop** as a result of increased calcium absorption from extracellular fluid of bone tissue. Treatment may require large doses of Ca i.v. . Surgical treatment is successful in 95% of cases.

Medical treatment is used in some cases of mild or subclinical course. **Estrogens, bisphosphonates, selective estrogen receptor modulators** and recently **calcimimetics** (e.g. cinacalcet) are used, which activate the calcium sensing receptor and lead to thereby reducing PTH secretion.

Severe hypercalcemia is life-threatening**.** Its treatment consists mainly of **hydration** and **forced diuresis.** Is it possible to i.v. administer **bisphosphonates** (pamidornate in a dose of 30–90 mg per day in an infusion of 0.9% NaCl solution) or the mentioned **calcimimetic**. In case of unsuccessful conservative treatment or life-threatening hypercalcemia, we choose **hemodialysis**.

Secondary hyperparathyroidism

It occurs as the **body's reaction to hypocalcemia.** So it is a physiological reaction. In the laboratory findings **low calcemia** and **high levels of parathyroid hormone** are found. There are many causes of secondary hyperparathyroidism, for example: **chronic renal failure, reduced absorption of vit. D due to intestinal inflammations, dietary deficiency of vit. D or calcium** etc.

Tertiary hyperparathyroidism

It arises during **long-term secondary hyperparathyroidism**, when there is hyperplasia of one or more bodies and autonomous secretion of PTH. Laboratory findings include **hypercalcemia** and **elevated PTH levels**.

Understanding Hyperparathyroidism - YouTube (https://www.youtube.com/watch?v=j_LPQT5t_88)

Odkazy

Související články

- Parathyroid glands
- Hypoparathyroidism
- Disorders of calcium phosphate metabolism

References

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