MEDICAL UNIVERSITY – PLEVEN

FACULTY OF MEDICINE - DISTANCE LEARNING CENTRE

DIVISION OF ENDOCRINOLOGY AND METABOLISM

Lecture №3

Parathyroid glands

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Disorders of the Parathyroid glands

Physiology

- **The function of the parathyroid glands is to regulate the amount of calcium in the body.**
- **The glands manufacture and secrete parathyroid hormone-PTH.**
- **If the calcium level in the body is low, more PTH is secreted.**
- **If the calcium level in the blood is too much the parathyroid glands decrease the level of PTH.**

Hyperparathyroidism

Hyperparathyroidism is overactivity of the parathyroid glands resulting in excess production of parathyroid hormone (PTH).

Classification

Primary

Excessive PTH secretion may be due to problems in the glands themselves which leads to hypercalcaemia.

Secondary

It occurs in response to low calcium levels, due to vitamin D deficiency or chronic kidney disease.

In both cases, the raised PTH levels are harmful to bone, and treatment is needed.

Primary Hyperparathyroidism

There is oversecretion of PTH due to
Adenoma - 80%,
Hyperplasia -rarely-15%,
carcinoma of the parathyroid glands 1-2%.

In a minority of cases this occurs as part of a
Multiple endocrine neoplasia (MEN) syndrome,

Secondary hyperparathyroidism

Is due to physiological (i.e. appropriate) secretion of parathyroid hormone (PTH) by the parathyroid glands in response to hypocalcemia .

The most common causes are vitamin D deficiency (caused by lack of sunlight, diet or malabsorption) and chronic renal failure.

Lack of vitamin D leads to reduced calcium absorption by the intestine causiang to hypocalcaemia and increased parathyroid hormone secretion. This increases bone resorption.

In chronic renal failure the problem is more specifically failure to convert vitamin D to its active form in the kidney.

The bone disease in state of the secondary parathyroidism caused by renal failure is termed renal osteodystrophy.

Tertiary hyperparathyroidism

Tertiary hyperparathyroidism is seen in patients with long-term secondary hyperparathyroidism which eventually leads to hyperplasia of the parathyroid glands and a loss of response to serum calcium levels.

This disorder is most often seen in patients with chronic renal failure and is an autonomous activity.

Symptoms and signs

Clasical symptoms:

1. **Renal disease symptoms – renal‘ stones, decreased renal function and nephrocalcinosis,**

2. **Classical hyperparathyroid bone disease – red-brown tumors**
3. **Gastric pain with nausea, vomiting and constipation.**

Parathyroid adenomas are very rarely detectable on clinical examination.

Symptoms and signs

In secondary hyperparathyroidism

The parathyroid gland is behaving normally; clinical problems are due to bone resorption and manifest as bone syndromes such as rickets, osteomalacia and renal osteodystrophy.

Laboratory tests

In primary hyperparathyroidism, PTH levels will be either elevated or "inappropriately normal" in the presence of elevated calcium.

Typically PTH levels vary greatly over time in the affected patient and (as with Ca and Ca++ levels) must be retested several times to see the pattern.

The currently accepted test for PTH is "Intact PTH" which is intended to detect only relatively intact and biologically active PTH molecules.

Older tests often detected other, inactive fragments.

Even "Intact PTH" may be inaccurate in patients with renal dysfunction.

Diagnosis

The gold standard of diagnosis is the PTH immunoassay. Once an elevated PTH has been confirmed, goal of diagnosis is to determine whether the hyperparathyroidism is primary or secondary

PTH	serum calcium	likely type
High	high	primary
High	low or normal	secondary

Treatment

Treatment depends on the type of hyperparathyroidism.

Patients with primary hyperparathyroidism who are symptomatic are candidate for surgery to remove the **parathyroid tumor (parathyroid adenoma).**

In patients with secondary hyperparathyroidism, the high PTH levels are an appropriate response to low calcium and treatment must be directed at the underlying cause of this (usually vitamin D deficiency or chronic renal failure).

If this is successful PTH levels should naturally return to normal levels unless PTH secretion has become autonomous (tertiary hyperparathyroidism)

Treatments of hypercalciemia

Initial therapy: fluids and diuretics

1. **Appropriate hydration**, increasing salt intake, and forced diuresis. **Hydration** is needed because many patients are dehydrated due to vomiting or renal defects in concentrating urine.
2. **Increased salt intake** also can increase body fluid volume as well as increasing urine sodium excretion, which further increases urinary calcium excretion

After rehydration, a **loop diuretic** such as furosemide can be given to permit **continued large volume intravenous salt and water replacement** while minimizing the **risk of blood volume overload** and **pulmonary oedema**.

Hypoparathyroidism

Hypoparathyroidism is the decreased function of the parathyroid glands with underproduction of parathyroid hormone.

Ethiology

- Inherited
- After thyroid or parathyroid gland surgery
- Autoimmune- it can be caused by immune system-related damage
- Other rarer causes

Signs and symptoms

The main symptoms of hypoparathyroidism are the result of the low blood calcium level, which interferes with normal muscle contraction and nerve conduction.

This can lead to cramping and twitching of muscles or tetany (involuntary muscle contraction) and several other symptoms.

- **Paresthesia**, an unpleasant tingling sensation around the mouth and in the hands and feet,
- **Muscle cramps** and severe spasms known as "tetany" that affect the hands and feet. Crampy abdominal pain may occur.

- **Fatigue,**
- **Headaches,**
- **Bone pain** and
- **Insomnia.**

Treatment

Severe hypocalcemia, a potentially life-threatening condition, is treated as soon as possible with **intravenous calcium** (**i.v. calcium gluconate 10%, 10 ml.).**

- **Calcium is administered by intravenous (IV) infusion in the event of a life-threatening attack of low calcium levels or tetany.**

Generally, a central venous catheter is recommended, as **the calcium can irritate peripheral veins and cause phlebitis**.

When the life-threatening attack has been controlled, treatment continues with medicine taken by mouth.

- **Long-term treatment of hypoparathyroidism is with vitamin D analogs and calcium supplementation**.