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Factors That Cause Treotoxicosis

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Abstract: what are the factors that cause treotoxicosis and how to eliminate it are studied.

Keywords: Diffuse toxic goiter, thyrotoxicos, hormon.

Thyrotoxicosis is a clinical syndrome that occurs with an increased content of thyroid hormones in the blood. An excess of these hormones affects the functioning of various organs and systems. Patients complain of dry skin, brittle hair, puffiness of the face, there may be a fine tremor of the eyelids, fingers and even the whole body. There are small fluctuations in temperature, increased nervous excitability, sweating, a feeling of heat, fussiness.

The patient develops swelling and darkening of the skin of the upper eyelids, lacrimation, photophobia, a feeling of pressure and "sand" in the eyes, bug-eyed (exophthalmos). With moderate or severe eye damage, there may be a decrease in visual acuity, patients cannot close their eyelids, therefore, corneal and scleral lesions develop (ulceration, infection).

Under the influence of provoking factors (stressful situations, physical overstrain, infectious diseases, surgical intervention), a thyrotoxic crisis may occur. As a result of the sudden release of a large amount of thyroid hormones into the blood, patients become restless, body temperature rises significantly, tachycardia increases sharply, breathing becomes faster, blood pressure increases. Types of thyrotoxicosis

I. Thyrotoxicosis caused by increased production of thyroid hormones:

1. Graves' disease (diffuse toxic goiter).

2. Multi-node toxic goiter.

3. Toxic thyroid adenoma.

4. Thyroid cancer.

5. TSH-secreting pituitary adenoma.

II. Thyrotoxicosis caused by the production of thyroid hormones outside the thyroid gland:

1. Chorionepithelioma.

2. Struma ovarii (ovarian tumor).

3. Functioning thyroid cancer metastases.

III. Thyrotoxicosis not associated with hyperproduction of thyroid hormones:

1. Medical thyrotoxicosis.

2. Thyrotoxic phase of destructive thyroiditis (subacute, postpartum). With a mild form of thyrotoxicosis, the pulse does not exceed 100 beats per minute, body weight loss is no more than 5 kg, eye symptoms are absent or slightly pronounced. With an average form - a pulse of

100-120 beats per minute, weight loss - 8-10 kg, pronounced tremor (trembling), increased systolic blood pressure and decreased diastolic pressure, exophthalmos. The severe form develops with long-existing thyrotoxicosis without treatment. The pulse rate is 120-140 beats per minute, weight loss reaches exhaustion, there is damage to the cardiovascular system, liver, adrenal glands.

An increase in thyroid hormones in the blood can be observed for several reasons:

- as a result of increased thyroid hormone production in the thyroid gland;
- as a result of the destruction of thyroid tissue and the ingestion of a large amount of thyroid hormones into the blood;
- in case of an overdose of thyroid hormone preparations or as a result of side effects of amio drug therapy.

Diffuse toxic goiter (Graves' disease) is the most common cause of increased thyroid hormone production. Depending on the nature of the enlargement of the thyroid gland, diffuse toxic goiter (diffuse enlargement of all parts of the gland) and nodular toxic goiter (focal enlargement of the thyroid gland) are distinguished.

Thyrotoxicosis can be observed against the background of subacute thyroiditis. Subacute thyroiditis (inflammation of thyroid tissue) occurs as a result of a viral disease and proceeds as a typical inflammation. In the period preceding the development of the disease, muscle pain, malaise, subfebrile fever, general weakness, sore throat, fatigue may be observed. Then there is moderate or severe pain in the thyroid gland, often radiating into the ears, jaw or throat. Sometimes there is pain when swallowing and turning the head. The thyroid gland is usually slightly enlarged, increasing fibrosis of the gland (proliferation of connective tissue) is manifested by an increase in its density There are 4 phases in the development of subacute thyroiditis. The first is thyrotoxic, which lasts from 4 to 10 weeks. It develops in the acute stage of the disease due to increased vascular permeability against the background of inflammation and increased release of previously synthesized thyroid hormones. Patients show symptoms of thyrotoxicosis. When the hormone reserves in the thyroid gland are depleted, the euthyroid phase begins, which lasts 1-3 weeks. It is replaced by the hypothyroid phase, which lasts from 2 to 6 months, and then recovery occurs.

The phenomena of thyrotoxicosis can manifest against the background of postpartum thyroiditis 1.5-3 months after delivery.

Chorionepithelioma is a malignant neoplasm that forms from chorionic epithelial cells during or after pregnancy more often in the uterus and produces the hormone chorionic gonadotropin. This hormone is a weak stimulator of thyroid cells. But at its high concentrations (300,000 units / l), thyrotoxicosis may occur. Struma ovarii, or ovarian tumor, refers to teratomas (tumors from atypical tissue for a given localization) in which thyroid tissue predominates or constitutes a significant component of the tumor. In 20-30% of cases, the tumor is represented only by thyroid tissue. 5-6% of these tumors produce thyroid hormones in an amount sufficient for the development of thyrotoxicosis.

Large hormone-active metastases of follicular thyroid cancer are very rare causes of thyrotoxicosis.

Thyrotoxicosis can occur with an overdose of thyroid hormones, due to inadequate iodine prophylaxis or against the background of the use of amiodarone.

Amiodarone is a highly effective and widely used antiarrhythmic drug in cardiology. Thyroid dysfunction is a frequent side effect of amiodarone due to either excessive uncontrolled hormone synthesis in response to iodine loading and/or destruction of the gland. Amiodarone-induced thyrotoxicosis is of great importance because it.

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