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DEPARTMENT OF INTERNAL DISEASES №2

DISEASES OF THE THYROID GLAND

METHODOLOGICAL MATERIALS
the main professional educational program of higher education - a program of a specialist in a specialty 31.05.01 General medicin

Vladikavkaz

Methodological materials are intended for teaching 5th year (9th semester) students of the medical faculty of the Federal State Budgetary Educational Institution of Higher Education SOGMA of the Ministry of Health of the Russian Federation in the discipline "Endocrinology".

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GOITER DIFFUSE TOXIC (Graves' disease).

Disease of an autoimmune nature, which is based on the hyperfunction of the thyroid gland, accompanied by its hyperplasia.

Etiology and pathogenesis.
Predisposing factors include:
1. Hereditary predisposition. Carriers of antigens of the HLA-B8, DR3, DR35 system are a genetic marker.
2. Foci of chronic infection, especially the tonsils.
3. The presence of other autoimmune diseases.
An auxiliary role in the etiology of diffuse toxic goiter (DTZ) is played by hormonal changes during puberty and during menopause, leading to an overstrain of the immune system.

Pathogenesis
DTZ is currently considered from the standpoint of an autoimmune disease. It has been proven that with DTZ there is a defect in the immune homeostasis system. Reduced suppressive activity of T-lymphocytes, which contributes to the accumulation of pathological clones of lymphocytes, which interact with organ-specific antigens of the thyroid gland. B-lymphocytes, producing thyroid-stimulating immunoglobulins, are also involved in the pathological process. They interact with receptors located on the membranes of thyroid cells and lead to an increase in the function of the thyroid gland, acting like thyrotropin. There is also the formation of antibodies to thyrotropin receptors.

Clinical picture...
Early signs characterized by emotional lability, sleep disturbance, sweating, palpitations, pain in the heart, trembling fingers, weight loss with increased appetite.
Stage of advanced clinical symptoms... Attention is drawn to an intense, anxious look, fussiness of movements, finely sweeping tremor of the fingers and the whole body. The skin is moist, elastic, there is a decrease in the turgor of the subcutaneous tissue. The color of the skin around the eyelids or the whole body may be dark. Sometimes there is a thickening of the skin in the area of \ u200b \ u200bthe feet and legs (pretibial myxedema). The thyroid gland is usually enlarged, diffuse on palpation, of varying density, mobile, painless.
Some patients have eye symptoms and manifestations of ophthalmopathy: wide opening of the eye slits (Delrimple symptom), glitter of the eyes, rare blinking (Stelwag symptom), lagging of the upper eyelid from the iris during downward movement of the eyeball with a fixed gaze behind a moving object (Gref's symptom), impaired convergence of the eyeballs (Mobius symptom), eyelid pigmentation (Jellinek symptom), exophthalmos, swelling of the eyelids, conjunctiva, impaired corneal trophism.

*Changes in the cardiovascular system*occupy one of the leading places in the DTZ clinic and can determine the prognosis of the disease. First of all, tachycardia attracts itself, which is characterized by stability at rest, even during sleep. Dysfunction of excitability is manifested by extrasystole and atrial fibrillation. Heart sounds on auscultation are sonorous, enhanced. Heart failure develops primarily in patients with atrial fibrillation due to increased load on the right ventricle. Systolic blood pressure is usually high, and diastolic blood pressure is low, which causes an increase in pulse pressure.

Disturbances from the digestive system are clinically manifested in the form of dyspeptic disorders: frequent loose stools, sometimes nausea and vomiting. An important prognostic clinical symptom of DTG is liver damage associated with the toxic effect of excess thyroid hormones. Patients may develop fatty degeneration of the liver.

Damage to the central nervous system manifested by insomnia, headache, dizziness. A common symptom of the disease is thyrotoxic myopathy, which is characterized by severe muscle weakness.

Involvement of other endocrine glands in the process accompanied by a violation of the menstrual cycle in women. Men may have decreased potency, gynecomastia. With the depletion of the capabilities of the adrenal cortical layer, hypocorticism develops.

By the nature of the flow, there are ***mild, moderate and severe form of DTZ.***

When ***mild form*** body weight loss does not exceed 10% of the initial, pulse pressure is not increased, tachycardia is moderate, not exceeding 100 beats per minute.

Moderate severity DTZ is characterized by a loss of body weight up to 20%, a significant increase in pulse pressure, pronounced tachycardia (more than 100 beats per minute). May be accompanied by myopathic syndrome.

Severe form DTZ is characterized by a significant loss of body weight, more than 20%, tachycardia of more than 120 beats per minute, atrial fibrillation, circulatory failure, hepatopathy, myopathy, often accompanied by ophthalmopathy.

Diagnosis and differential diagnosis.

Diagnosis based on the analysis of clinical signs (weight loss with good appetite, emotional lability, stable tachycardia, increased pulse blood pressure, thyroid hyperplasia, eye symptoms, tremors of the hands and the whole body). Difficulties in making a diagnosis may be associated with a multisymptomatic course of the disease or the predominance of one of the symptom complexes. As a rule, with a detailed clinical picture of DTG, the diagnosis is not difficult. In the case of initial and mild forms of this disease, the following criteria are of diagnostic value:

- an increase in the content of T4 and T3 in the blood serum (T4 more than 140 nmol / l, T3 more than 2.0 nmol / l);
- an increase in the level of thyroglobulin in the blood serum, a decrease in the level of TSH less than 0.6 mIU / l;
- on the ECG: sinus tachycardia, sinus arrhythmia, high voltage of the teeth, atrial fibrillation, biphasic or negative T wave;
- shortening of the Achilles reflex time less than 180 m / s (230-270 m / s);
- in the biochemical analysis of blood: hypocholesterolemia, hypoalbuminemia, hyperbilirubinemia, increased levels of transaminases;
- in the general analysis of blood, there may be leukopenia, absolute or relative lymphocytosis, relative or absolute neutropenia, thrombocytopenia, anemia;
- changes in the immunogram: from the humoral link of immunity - the presence of thyroid-stimulating antibodies in the blood serum, antibodies to thyroglobulin;
- on the part of the cellular link of immunity: a decrease in the number of total T-lymphocytes, a decrease in T-suppressors, an increase in the number of activated T-lymphocytes.

On ultrasound, there is an increase in the size of the thyroid gland, uneven changes in echogenicity with foci of its decrease.

Depending on the leading clinical syndrome of DTZ, it is necessary ***differentiate*** from the following diseases:

1. Neurocirculatory dystonia and climacteric neurosis.
2. Rheumatic heart disease and rheumatic heart disease.
3. Atherosclerotic cardiosclerosis and aortic atherosclerosis.
4. Chronic enterocolitis.
5. Tuberculosis.
6. Malignant neoplasm of the thyroid gland.

Common signs of neurocirculatory dystonia and DTG there may be emotional lability, sweating, tachycardia, a tendency to arterial hypertension.

The difference DTZ consists in progressive weight loss, stable tachycardia, even at rest, during sleep. An increase in pulse pressure, the presence of eye symptoms and an enlargement of the thyroid gland, along with indicators of additional diagnostic methods (high levels of T4, T3, low plasma cholesterol).

General clinical symptoms of climacteric neurosis and DTG are: irritability, poor sleep, tearfulness, feeling of heat, increased sweating.

The difference DTZ consists in increasing weakness and weight loss, while in the climacteric period there is a tendency to increase body weight, in addition, the feeling of heat in patients with DTZ is constant, and in menopause it is noted as "hot flashes", replaced by a feeling of chilliness. Additional research methods help formulate the correct diagnosis.

Common signs of rheumatic heart disease and DTG: low-grade body temperature, palpitations, shortness of breath, general weakness, pain in the heart.

The difference DTZ in the stability of tachycardia, sonorous, intensified heart sounds, progressive weight loss with good appetite, as well as in the presence of eye symptoms and an enlarged thyroid gland.

Differential diagnosis with aortic atherosclerosis carried out according to the general symptom of an increase in pulse pressure in cases of mild clinical signs of thyrotoxicosis in elderly and senile people.

The difference DTZ consists in the stability of tachycardia, which cannot be treated with glycosides, emotional lability, a tendency to sweating, in a decrease in blood cholesterol and an increase in the content of thyroid hormones.

Treatment.

Drug therapy for DTZ is the main method of treatment and is used until the symptoms of DTZ are completely eliminated. Complex drug therapy of DTZ includes the appointment of thyreostatics, beta-blockers, synthetic thyroid hormones, immunocorrectors, hepatotropic and anabolic drugs.

For thyrostatic therapy, propylthiouracil, tyrosol, thiamazole, and mercazolil are most effective. At the beginning of treatment, the optimal therapeutic doses are used, which are gradually reduced under the control of clinical symptoms and the level of T3 and T4 in the blood. The listed drugs block the synthesis of T3 and T4 at the level of iodinated tyrosines, which leads to

a decrease in the thyroid hormones circulating in the blood and helps to reduce the symptoms of thyrotoxicosis.

The criterion for dose reduction are: normalization of body weight, stabilization of blood pressure, elimination of autonomic reactions. Treatment with a maintenance dose is carried out for at least 12 months.

When prescribing thyreostatics, leukopenia may develop, therefore, it is necessary to do a general blood test 1 time in 7-14 days. In addition, nausea, skin rash, and joint pain are possible. With the development of side effects, the dose of mercazolil is reduced or further administration of the drug is stopped.

For the treatment of DTZ in cases of intolerance to these drugs, lithium carbonate can be used, 0.3 g 2-3 times a day.

Complex therapy of DTZ includes the use of beta-adrenergic receptor blockers. This leads to a decrease in the manifestations of thyrotoxicosis. It is advisable to use these drugs simultaneously with thyreostatics. The average dose of beta-blockers (anaprilin, obzidan, trazikor) is 40-120 mg / day. After reaching the euthyroid state, the dose is gradually reduced. The maintenance dose ranges from 20 to 40 mg / day.

In addition to thyrostatics and beta-adrenergic receptor blockers, thyroid hormones are used to prevent drug-induced hypothyroidism.

Immunocorrective therapy is aimed at suppressing the formation of autoantibodies and normalizing cellular immunity factors. It is an essential component of successful drug therapy. The disappearance of thyroid-stimulating antibodies in the blood serum of patients with DTG against the background of the euthyroid state indicates the cessation of autoaggression.

To achieve clinical and immunological remission, along with thyreostatics with immunocorrective properties, immunosuppressants (glucocorticoid drugs), immunomimetics, and methods of gravitational surgery are used. In the treatment of DTG, preference should be given to synthetic drugs of glucocorticoid action (prednisolone, dexamethasone, kenacort, etc.). Prednisolone is prescribed at 20-40 mg / day in courses of 1 month. 2-4 such courses are recommended. The therapeutic complex for DTZ includes antihistamines (tavegil, suprastin, diazolin), antiserotonin drugs (cyproheptadine, peritol). For the prevention of thyrostatic hepatosis, hepatoprotectors are used (legalon, carsil, essential). In case of heart failure, it is recommended to add cardiac glycosides to the treatment, the appearance of extrasystole or atrial fibrillation is an indication for the appointment of antiarrhythmic therapy.

Indications for surgical treatment: lack of clinical compensation against the background of correct therapy for 6-8 months; absence of clinical and immunological remission in case of withdrawal of thyrostatic drugs within 2 years; large size of the thyroid gland with signs of compression of the neck organs, not amenable to drug correction; manifestation of DTZ during pregnancy. It should be remembered that surgical treatment can be carried out only against the background of clinical and hormonal compensation of thyrotoxicosis.

Treatment with radioactive iodine can be recommended for people with severe and complicated form of DTZ and the presence of concomitant diseases that prevent surgical treatment; with relapses of DTG after surgical treatment; in case of refusal of the patient from the operation with the ineffectiveness of complex drug therapy.

Hypothyroidism

Hypothyroidism is a group of diseases that are caused by insufficient thyroid function.

Etiology and pathogenesis...

Depending on the etiological and pathogenetic factors, congenital and acquired hypothyroidism is distinguished, as well as primary (with damage to the thyroid gland), secondary (with insufficient secretion of thyrotropin), tertiary (due to deficiency of thyroliberin) and peripheral (with a decrease in the sensitivity of cells of organs and tissues to thyroxine and triiodothyronine)

The causes of congenital hypothyroidism are aplasia (hypoplasia) of the thyroid gland and genetically determined disorders of the biosynthesis of thyroid hormones.

Causes of acquired hypothyroidism:

1) primary - strumectomy, ionizing irradiation of the thyroid gland (with X-ray therapy of the neck organs, treatment with radioactive iodine, the action of iodine radionuclides from the environment), autoimmune thyroiditis, endemic goiter,

drugs (imidazole derivatives, lithium preparations, glucocortacoids, beta-blockers adrenergic receptors, iodides);
2) secondary - a decrease in thyrotropin secretion due to hemorrhages in the adenohypophysis, inflammatory processes, tumors, as well as under the influence of drugs (reserpine, parlodel, apomorphine);
3) tertiary — insufficient secretion of thyroliberin after viral diseases, with head injuries and brain tumors, under the influence of drugs containing serotonin;
4) peripheral - inactivation of thyroid hormones during circulation or a decrease in the sensitivity of the receptors of thyroid-dependent cells to the action of thyroxine and triiodothyronine. This form is the least studied and difficult to treat.

Pathogenesis

In hypothyroidism, pathogenesis is caused by a deficiency of thyroid hormones that regulate the growth and development of the body and its energy metabolism.
Disorders of protein metabolism are characterized by a slowdown in both biosynthesis and decay, accumulation of hyaluronic, chondroitinsulfuric acids and mucin glycoprotein in tissues, which causes mucous edema of tissues and organs, contributing to the development of hydrothorax, hydropericardium.
Shifts in lipid metabolism are associated with a decrease in utilization and a slowdown in decay, which contributes to an increase in the concentration of cholesterol, triglycerides, beta-lipoproteins in the blood.
Disorders of carbohydrate metabolism consist in impairing the absorption of glucose in the intestine and slowing down its utilization.
In primary hypothyroidism, thyrotropin secretion increases, and in secondary and tertiary hypothyroidism, it is reduced. The secretion of thyroliberin in primary hypothyroidism is also increased, while the secretion of prolactin also increases.

The clinical picture.

Early symptoms hypothyroidism is not very specific, and therefore the initial stages of the disease, as a rule, are not recognized and patients are unsuccessfully treated for "depression", "anemia", "cardiovascular disorders", "kidney disease".
The initial signs of hypothyroidism include a feeling of chilliness, unmotivated weight gain, slowness, daytime sleepiness, dry skin, subcutaneous tissue density, hypothermia, a tendency to bradycardia, constipation. Galactorrhea is sometimes the first symptom of primary hypothyroidism (Van Wyck-Hennes-Ross syndrome). There may be symptoms of polyneuritis (pain, sensory disturbances, paresthesia) or radiculitis.
Stage of advanced clinical symptoms*:* subjective sensations are characterized by memory loss, difficulty speaking, muscle weakness. An objective examination reveals puffiness of the face, pasty eyelids, lethargy, indifferent gaze, and slowness of movements. The patient has a low voice, slow speech, the skin is cold, yellowish, dense to the touch, does not gather in folds, in the area of the elbows there is peeling. Swelling of the mucous membranes leads to an increase in the tongue, often teeth imprints are visible on it.

The pathogenesis of edematous syndrome consists in the deposition of mucin and glucosaminoglycans, which are hydrophilic, prone to lymphastasis. Thinning of hair on the head and loss of the outer third of the eyebrows (Hertog's symptom) are observed. There may be ophthalmopathy, especially in the autoimmune genesis of hypothyroidism.

Changes in the cardiovascular system are manifested by bradycardia, arterial hypotension, weakening of the apical impulse, deafness of heart sounds, which is associated with myocardial dystrophy and weakening of its contractile function. Changes in hemodynamics are characterized by a decrease in stroke and minute blood volume, as well as the volume of circulating blood. Often with hypothyroidism, arterial hypertension is also noted. The deposition of glucosaminoglycans in the mucous membrane of the nasopharynx leads to difficulty in nasal breathing and the development of vasomotor rhinitis. Anorexia, a decrease in the secretion of enzymes of the stomach and small intestine, a violation of the motor function of the biliary tract are noted. Decreased tone and motor function of the colon sometimes resembles intestinal obstruction syndrome. The reabsorption and secretory functions of the kidneys decrease, which is manifested by a decrease in urine output.

Changes in the central and peripheral nervous system play a significant role in the clinical picture of hypothyroidism. Disorders of the peripheral nervous system are characterized by polyneuritis syndrome or clinical symptoms of radiculitis, and the central nervous system by depression, memory loss, apathy, withdrawal, distraction, hypochondria. They often lead to a decrease in working capacity, especially in persons engaged in mental work.

Patients have factors such as anemia and increased blood clotting.

Diagnosis and differential diagnosis...

It is important to recognize the subclinical stages of hypothyroidism.

One of the reliable tests for early diagnosis of hypothyroidism is a decrease in the index of free thyroxine (Tl) in blood plasma (N - 50-110 nmol / l), an increase in thyrotropin levels (N - 0.5-3.5 mIU / l). With hypothyroidism, the indicator of the binding ability of blood proteins to thyroid hormones increases. There is a binding of thyroxine by immunoglobulins G and M. The test with thyroliberin (500 μg intravenously) in primary hypothyroidism is accompanied by an increase in the level of thyrotropin, while the content of T3 and T4 does not change in response to stimulation. The test with thyroliberin is especially important for the diagnosis of subclinical stages of primary hypothyroidism. An auxiliary diagnostic value is the determination of the level of cholesterol and beta-lipoproteins in the blood serum, the content of which is increased in hypothyroidism. The hemogram is characterized by normochromic anemia, lymphocytosis, leukopenia. In connection with dysproteinemia, ESR may increase. The ECG shows a low voltage of the main teeth, the R - R interval increases, the R - T interval lengthens, the T wave is flattened or its weak inversion is observed, the electrical axis of the heart deviates to the left, the electrical position of the heart approaches horizontal. A certain

diagnostic value is assigned to reflexometry (lengthening of the reflex from the Achilles tendon more than 300 m / s).

Depending on the leading clinical syndrome, hypothyroidism must be differentiated from the following diseases: chronic ischemic heart disease (IHD) with circulatory failure; chronic glomerulonephritis with kidney failure; anemia.

Common signs for hypothyroidism and chronic coronary heart disease - shortness of breath, edema of the subcutaneous tissue, hypercholesterolemia, decrease or inversion of the T wave on the ECG. The difference between hypothyroidism and ischemic heart disease is that edema in hypothyroidism does not leave a trace when pressed, with hypothyroidism, bradycardia is noted, and in chronic heart disease with circulatory failure, the heart rate increases. On the ECG with hypothyroidism, there is no discordance of changes in the terminal part of the ventricular complex in the right and left chest leads, which is characteristic of chronic ischemic heart disease. There are also no signs of left ventricular hypertrophy on the ECG, no effect of treatment with cardiac glycosides is observed.

Common signs characteristic of hypothyroidism and chronic glomerulonephritis with impaired nitrogen-excreting renal function - swelling of the subcutaneous tissue, pasty and puffy face, pallor of the skin, anemia, increased ESR.

The difference -with hypothyroidism, lethargy and anemia, which is not characteristic of glomerulonephritis, are noted, the density of edema, changes in the timbre of the voice, arterial hypotension. With hypothyroidism, there is no protein, casts, or erythrocytes in the urinary sediment. The content of creatinine and urea in the blood plasma is not increased.

With the appointment of thyroid hormones, hypothyroidism is characterized by regression of symptoms.

Common signs of hypothyroidism and anemiaare pallor of the skin, a decrease in the number of red blood cells and a decrease in the level of hemoglobin in the blood. The difference lies in psychomotor retardation, characteristic of hypothyroidism, bradycardia.

Hypothyroidism is rigid to treatment with antianemic drugs, but the blood picture is restored with the appointment of thyroxine, triiodothyronine or thyrotome.

Treatment.

Adults.In hypothyroidism, regardless of the etiology of the disease, thyroid hormone preparations (triiodothyronine, L-thyroxine, eutirox, thyrotome) are prescribed, which eliminate the clinical manifestations of hypothyroidism. At the beginning of therapy, small doses of drugs are used: L-thyroxine and eutirox - 25-50 mcg / day.

Every 3-5 days, the dose is increased, bringing it to the optimum, allowing you to maintain the euthyroid state. The adequacy of the dose of thyroid hormones is assessed by the disappearance of the clinical symptoms of hypothyroidism based on the data of the biochemical blood test (normalization of lipid profile,

proteinogram), normalization of thyroxine, triiodothyronine and blood thyrotropin levels and the Achilles reflex time.

In addition to substitution therapy in the complex treatment of hypothyroidism are used: lipotropic drugs (lipostabil, lipoic acid), courses of 1 month, 1-2 courses during the year; peroxide blockers
lipid oxidation (aevit, alpha-tocopherol), 1 month 2 times a year; with severe edema, diuretics are prescribed (veroshpiron; triampur, uregid, furosemide, brinaldix in combination with potassium preparations); to improve liver function, hepatotropic drugs (Essentiale, Carsil, etc.) are used for 1 month, 2-3 courses per year.

Treatment of secondary and tertiary hypothyroidism is carried out taking into account the nature of the disease of the hypothalamic-pituitary system, which led to the development of hypothyroidism. For this purpose, anti-inflammatory treatment, radiation therapy to the hypothalamic-pituitary region, or surgical treatment of a pituitary adenoma can be used. In all cases of secondary or tertiary hypothyroidism, thyroid hormones are required to correct the thyroid status.

Elderly and senile age...Due to the activation of metabolic processes and an increase in myocardial oxygen demand while taking thyroid hormones, the treatment of patients with atherosclerosis of the coronary arteries with large doses may be accompanied by signs of coronary insufficiency, therefore, the initial dose of L-thyroxine for persons over 50 should not exceed 25 μg. The dose should be increased no earlier than after 1-2 weeks by 12.5-25 mcg, gradually bringing it to the optimal level within 2-3 months. Control of the adequacy of the dose in old age is the elimination of clinical signs of hypothyroidism. The approximate dose is 150 μg of L-thyroxine or eutirox per day. In this case, the T4 level may be at the lower limit of the norm. In patients who have had myocardial infarction, as well as those suffering from clinically severe coronary artery disease, it is inappropriate to completely eliminate the symptoms of hypothyroidism, because. due to the reduced oxygen demand against the background of hypothyroidism, angina attacks develop less frequently, and the risk of myocardial infarction decreases. At the same time, treatment with thyroid hormones in this category of patients can be combined with the use of coronary artery disease and drugs that reduce myocardial oxygen demand for the prevention of angina pectoris.

Newborns.Hypothyroidism of newborns can be caused by aplasia or hypoplasia of the thyroid gland, a deficiency of enzymes involved in the biosynthesis of thyroid hormones, as well as the intake of a pregnant strumogen, a deficiency or excess of iodine during intrauterine development. The reason may be the effect of radionuclides
fetus, since from 10-12 weeks of intrauterine development, the thyroid gland
the fetus begins to accumulate radioactive iodine.

Diagnostic criteria:

1. Clinical*:*large body weight of a newborn; swelling of the hands, feet, face, thick skin; hypothermia; lethargy; weak sucking reflex; intense weight gain.

2. Laboratory - screening of newborns with the determination of TSH on the 4-5th day using a test - a strip "neonatal", on which a drop of blood is applied from the heel.

An increase in TSH levels is an indication for thyroid hormone treatment. Treatment begins no later than 5-17 days after birth. If hypothyroidism is combined with chronic insufficiency of the adrenal cortex, first of all, correction with corticosteroids and careful selection of the dose of thyroid hormones are necessary to avoid an adrenal crisis.

Pregnant women*...*The key to a successful pregnancy can only be sufficient compensation for hypothyroidism. During pregnancy, the dose of thyroid hormones is increased under the control of the optimal content of T4 and T3 in the blood, as well as the normalization of TSH. Thyroid hormones do not have a teratogenic effect, since they do not penetrate the placental barrier. In this regard, during pregnancy and breastfeeding, a woman should take a sufficient dose of the medication.

Subacute thyroiditis (de KERVEN's thyroiditis).

A disease characterized by an inflammatory process in the thyroid gland is believed to be of viral etiology, accompanied by destruction of thyrocytes.

Etiology and pathogenesis.

The predisposing factors can be divided into genetic and environmental factors.

The genetic predisposition to the disease is associated with the features of the HLA histocompatibility system. Carriage of HLA-BW 35, DR 35 antigens is considered as a risk factor for the development of subacute thyroiditis under the action of predisposing environmental factors.

As an environmental etiological factor, a viral infection is most likely. In this case, the specific virus that causes the disease has not been established. Subacute thyroiditis can develop under the influence of the Coxsackie virus, adenoviruses, mumps virus, measles.

The following facts can serve as confirmation of the viral etiology of the disease:

- anamnestic relationship with a previous viral infection;
- an increase in the number of cases during periods of an outbreak of viral infections;
- the presence of high titers of antibodies to the Coxsackie virus, adenoviruses, influenza viruses in patients.

Early stage characterized by exudation, hyperemia, vasodilation, edema, swelling of epithelial cells.

Next stage - morphologically manifested by rupture of follicles and the entry of colloid and structural elements into the perifollicular space. Infiltration with lymphocytes, macrophages, giant cells is accompanied by the formation of

antibodies to thyroid antigens, which entered the bloodstream as a result of the destruction of thyrocytes. In this phase of the development of the disease, the morphological process corresponds to the autoimmune one, but the immune responses are secondary, because they develop in response to the primary effect of a virus damaging factor on thyrocytes. In this case, the immune changes may be temporary.

The third stage of the pathogenetic process is characterized by scarring.

This is the final stage of morphological changes.

Immunological aspects. The destruction of thyrocytes, which develops in the second stage of the pathological process, is accompanied by the entry into the circulation of thyroid autoantigens: colloid, microsomal fraction, nuclear component, thyroglobulin. The response is characterized by the formation of autoantibodies in relation to the listed components of the thyrocyte. The predisposition to them is associated with the presence of histocompatibility antigens. Viral antigens are complemented with histocompatibility antigens on the surface of thyrocytes, contributing to the progression of the pathological process.

Clinical picture...

The disease can begin acutely, with a rise in body temperature to febrile and pain in the neck, or gradually, accompanied by malaise, discomfort when swallowing, pain when turning or tilting the head.

Early signs: discomfort when swallowing, general malaise, soreness in the thyroid gland.

Stage of advanced clinical symptomscharacterized by intense pain in the neck with irradiation to the ears, lower jaw, occipital region. The pain is worse when turning the head, when chewing food, while swallowing. Intense sweating, palpitations, insomnia, arthralgia are noted.

Important information is given by the results of palpation of the thyroid gland, which is moderately enlarged, dense, painful; areas of local pain can also be determined. In the majority of patients in the initial period of the disease, symptoms of thyrotoxicosis are observed. General hyperhidrosis, expansion of peripheral blood vessels of the skin, tachycardia, symmetrical small-sweeping tremor of outstretched fingers. The development of these symptoms is due to the destruction of thyroid cells as a result of a pathological process, which leads to the ingress of hormones from damaged follicles into the blood.

Features of the clinical course:

By stages*:*

1. Stage of hyperthyroidism (at the onset of the disease).
2. The stage of hypothyroidism (coincides with the period of destructive-sclerotic changes in the thyroid gland).

By the prevalence of clinical symptoms:

1 A form with a pronounced inflammatory reaction.

2 Slowly progressive form, similar to chronic thyroiditis.

3 Form with clinically pronounced hyperthyroidism.

4 Pseudoneoplastic form.

Diagnosis and differential diagnosis. Diagnostic criteria:

***1 Etiological*:**the development of the disease during the period of recovery from previous viral infections; carriage of HLA antigens - B 35, DR35.

2 **Clinical:**acute onset of the disease with fever, neck pain, characteristic irradiation of pain (in the ear, back of the head), dysphagia; diffuse enlargement of the thyroid gland with local, very painful lumps, no enlargement of regional lymph nodes; recurrent course of the disease; manifestations of hyperthyroidism at the onset of the disease, increased pulse blood pressure, symmetrical tremor of outstretched arms, tachycardia, muscle weakness, emotional lability.

3 **Laboratory:**complete blood count - acceleration of ESR, lymphocytosis; nonspecific markers of an acute inflammatory reaction: positive CRP, increased blood levels of alpha 2 -globulins, fibrinogen; an increase in serum T3, T4, thyroglobulin with a reduced inclusion of radioactive iodine by the thyroid gland; there may be a temporary increase in the titer of antithyroid antibodies.

Differential diagnosis of subacute thyroiditis carried out with autoimmune thyroiditis.

Common signs: discomfort when swallowing, a feeling of pressure in the back of the pharynx, an increase in the size of the thyroid gland on palpation.

***Differences*:**with subacute thyroiditis, it is usually possible to identify a clear connection with an acute viral infection (influenza, measles, etc.), vivid clinical symptoms (fever, malaise); in the analysis of blood - a sharply increased ESR. Ultrasound examination in patients with autoimmune thyroiditis reveals the presence of local areas of reduced echogenicity, single or multiple. With subacute de Quervain's thyroiditis, a slight uniform decrease in echogenicity is usually detected,

grasping at least 1/3 of the thyroid gland. In some cases, for the purpose of differential diagnosis of these two diseases, they resort to a more informative method - fine-needle aspiration biopsy followed by cytological examination of the biopsy specimen.

The presence of symptoms of thyrotoxicosis in some patients with subacute de Quervain's thyroiditis dictates the need for differential diagnosis with diffuse toxic goiter.

***Common signs*:** sweating, tachycardia, shallow tremors of the hands, muscle weakness, enlarged thyroid gland and increased blood T4 levels.

Differences in diffuse toxic goiter consist in the progression of symptoms of thyrotoxicosis, diffuse enlargement of the thyroid gland and the absence of pain on palpation, in normal ESR values.

It should be noted that a clear clinical picture of thyrotoxicosis is observed only in the case of a severe course of subacute thyroiditis with severe malaise, fever, chills, severe pain when swallowing, tenderness on palpation of the thyroid gland, which is not typical for diffuse toxic goiter.

Treatment.
The main method of treatment is the appointment of synthetic glucocorticoid drugs (prednisone, dexamethasone, metipred). Prednisolone is prescribed at 40-50 mg / day, dexamethasone - 4-5 mg / day, respectively, according to the method of imitating physiological secretion or by an alternating method (at a 48-hour dose 1 time in the morning every other day). In mild cases of thyroiditis, salicylates (aspirin 0.5 g 3 times a day) in combination with a non-steroidal anti-inflammatory drug can be prescribed. Positive results were obtained with the introduction of steroid hormones directly into the thyroid gland by the puncture method (30 mg once a week). The administration of sodium nucleinate at a dose of 1.0 g per day is also very effective.
With pronounced manifestations of hyperthyroidism at the onset of the disease, it is advisable to prescribe adrenolytics.
After the elimination of the clinical symptoms of the disease and the normalization of ESR, the dose of anti-inflammatory drugs and steroid hormones is reduced. During this period, it is advisable to prescribe thyroid hormones, which can be gradually canceled after 1-1.5 months.

Forecast.
With timely diagnosis and adequate treatment, the disease ends with recovery within 2 to 3 months; in the absence of proper therapy, the duration of the disease can be up to 2 years, while undulation is noted in its course. Development of latent or clinical hypothyroidism is possible.

Thyroiditis autoimmune
(Hashimoto's thyroiditis, lymphomatous thyroiditis).

The disease, which is based on the autoimmunization of the body with thyroid autoantigens, followed by the production of autoantibodies and cytotoxic lymphocytes.

Etiology and pathogenesis...
The most common autoimmune thyroiditis occurs in women. The female to male ratio is approximately 15: 1. In recent years, clinicians in many countries have noted an increase in the number of patients with autoimmune thyroiditis among young people, especially among children and adolescents.
Genetic predisposing factors...
When studying the HLA system, a high frequency of carriage of antigens HLA - Bg, HLA - DRa, HLA - DRa was established in patients with autoimmune thyroiditis. Autoimmune thyroiditis is more often recorded in families where there are already patients with autoimmune endocrine and non-endocrine diseases (type 1 diabetes mellitus, diffuse toxic goiter, chronic adrenal insufficiency of autoimmune genesis, pretibial myxedema, vitiligo, endocrine diseases, rheumatic diseases respiratory tract).

The decisive factors contributing to the implementation of a genetic defect in the immunological control system can be respiratory diseases, chronic inflammatory processes of the nasopharynx and palatine tonsils, long-term intake of large doses of iodine-containing drugs, frequent X-ray examinations using contrast agents containing iodine, the effect of ionizing radiation.

Immunological aspects of pathogenesis are characterized by disorders in the immunological control system. Under the action of genetic (HLA) and environmental (chronic infection) factors, prohibited (phorbid) clones of T-lymphocytes are activated, which suppress the population of T-suppressors, which is accompanied by the formation of immunoglobulins, especially class G, which are the source of antibody production. Immunoglobulins complex with a subpopulation of killer T-lymphocytes and form cyto-damaging complexes that cause destruction of thyrocytes. As a result, the contents of follicles (a lot of thyroglobulin, microsomal fraction, colloid, nuclear components), which have antigenic properties, enter the bloodstream. In relation to them, autoantibodies are formed, and the process becomes cyclical.

Enzymatic defects in the biosynthesis of thyroid hormones are also involved in the pathogenesis of autoimmune thyroiditis.

The morphological aspects of pathogenesis are characterized by the presence of areas of infiltration by plasma cells, macrophages and lymphocytes. It should be noted the accumulation of lymphoid elements with varying degrees of differentiation. A special place is occupied by the so-called. Ashkenazi cells, the presence of which is characteristic of autoimmune thyroiditis. Gradually, as the processes of lymphoid infiltration spread, the number of functioning thyrocytes decreases and hypothyroidism develops.

Clinical picture...

During autoimmune thyroiditis, a long asymptomatic period of the disease is possible.

Early signs',a feeling of discomfort when swallowing, "a feeling of pressure in the throat", sometimes - slight pain in the thyroid gland on palpation. There may be arthralgias without signs of inflammatory changes in the joints. In general, the complaints of patients with autoimmune thyroiditis differ in their non-specificity, variety and transient nature of subjective sensations. Features of complaints are largely determined by the functional state of the thyroid gland at the time of examination (hyperthyroidism, thyrotoxicosis, euthyroidism or hypothyroidism). Symptoms of hyperthyroidism or thyrotoxicosis are more often recorded at the onset of the disease. Sweating, tachycardia, hand tremors, arterial hypertension appear. Further, states of euthyroidism or hypothyroidism (including subclinical) can be observed, sometimes hyperthyroidism again. The development of persistent hypothyroidism is observed in the late stages of autoimmune thyroiditis (from 5 to 15 years or more). Relapses of hyperthyroidism are provoked by acute respiratory infections, mental and physical overload, and in women - by pregnancy, childbirth, abortion. As the

disease progresses, there is a feeling of bloating in the eyeballs, persistent headaches. With an increase in the size of the thyroid gland (more than III degree), symptoms of compression of the neck organs (dysphagia) may develop.

Often, autoimmune thyroiditis is combined with endocrine ophthalmopathy, pretibial myxedema. When assessing the objective status of patients, one should remember about the possible clinical manifestations of adrenal cortex insufficiency, insulin-dependent diabetes mellitus.

By the nature of changes in the thyroid gland, atrophy is distinguished
i'sand hypertrophic forms of autoimmune thyroiditis. In turn, the hypertrophic form can be diffuse, diffuse-nodular and nodular.

In the atrophic form, which is more common in the elderly, there is no increase in the size of the thyroid gland. However, there is a significant condensation. The atrophic form can be in young people, especially if. the process is due to the action of the radiation factor.

Hypertrophic diffuse formcharacterized by an increase in the thyroid gland of varying degrees (from I to III), density and sometimes sensitivity to palpation. With a diffuse nodular form, there is a different density of nodular formations against the background of a diffusely enlarged thyroid gland. The nodular form resembles a nodular goiter or adenoma and is characterized by the presence of a dense, mobile nodular formation in the region of the lobe or isthmus. With autoimmune thyroiditis, there may be an increase in regional lymph glands.

Diagnosis and differential diagnosis...

Diagnostic criteria: etiopathogenetic - carriage of HLA antigens: DRs, DR3, Bg; the presence of concomitant endocrine and non-endocrine diseases; exposure to small doses of radionuclides; excessive intake of iodine, heavy metal salts, pesticides, herbicides into the body.

Clinical - the presence of a goiter of pronounced density with a hypertrophic form, an increase in regional lymph nodes, signs of hyperthyroidism or hypothyroidism, depending on the stage of the pathological process.

Laboratory:in the general analysis of blood - lymphocytosis, monocytosis, leukopenia; radioimmunological study of hormonal status: at the stage of hyperthyroidism - increased blood levels of T3, T4; with a decrease in the function of the thyroid gland, the level of thyrotropin increases (the earliest sign of hypothyroidism), at the same time T4 decreases, to a lesser extent - T3. The development of hypothyroidism in the early stages can be confirmed by a test with thyroliberin (200 μg intravenously) - a sharp rise in the level of thyroid-stimulating hormone 30 minutes after the administration of the drug (two or more times higher than the physiological response of thyroid-stimulating hormone to the pharmacological load).

In the immunogram from the side of the cellular link, an increase in the number and activity of T-helpers and killers is noted with a decrease in the number
T-suppressors. Changes on the part of the humoral link are characterized by an increase in the content of immunoglobulins, mainly of class G, complement

components and complement titer. Antibodies to the microsomal fraction, nuclear antigens, colloid appear, and the titer of antibodies to thyroglobulin sharply increases.
With ultrasound sonography, there is an increase in the size of the lobes and isthmus with a hypertrophic form, an uneven picture with the participation of normal, increased and reduced echogenicity (mosaic).
On the scanned image, there is a variegation of the image due to the alternation of areas with active absorption of the isotope and areas that weakly and do not absorb the isotope at all.
Cytological examination of punctate obtained with fine-needle aspiration biopsy reveals lymphoid cells at different stages of differentiation, Ashkenazi cells, and plasma cells.

Differential diagnosis...

Differentiate autoimmune thyroiditis with nodular euthyroid goiter, thyroid cancer, diffuse toxic goiter, Riedel's fibrous thyroiditis.
Nodular euthyroid goiter... *Common signs:* the presence of a dense node in the area of the lobe or isthmus of the thyroid gland.
Differences in nodular goiter - the absence of signs of autoaggression, in the punctate of the thyroid gland there is no lymphoplasmacytic infiltration, in the blood serum there are no autoantibodies to the structural components of the follicles (microsomal fraction, colloid, nuclear component).
Thyroid cancer... *Common signs:* nodules, thyroid density and regional lymph node enlargement.
Differences:in cancer, the nodular formations are inactive, their adhesion with the surrounding tissues is noted. Puncture biopsy reveals undifferentiated cells with signs of proliferation.
Diffuse toxic goiter... *Common signs:* symptoms of thyrotoxicosis, which can be observed in the early stages of the autoimmune process when excess thyroid hormones enter the bloodstream.
Differences*:* no progression of thyrotoxicosis without the use of thyrostatic therapy with self-restoration of euthyroid status in autoimmune thyroiditis.
Riedel's fibrous thyroiditis... *Common signs:* enlargement and induration of the thyroid gland, discomfort when swallowing.
Differences*:* with fibrous Riedel thyroiditis - "woody" dense
The thyroid gland is fused with the surrounding tissues, there are no symptoms of thyrotoxicosis or hypothyroidism.

Treatment.

Complex therapy of autoimmune thyroiditis depends on its form (atrophic or hypertrophic, diffuse-nodular), the functional state of the thyroid gland, and the severity of autoaggression. The main method of treatment is medication. It is generally accepted to prescribe synthetic thyroid hormones (triiodothyronine, levothyroxine) for a long time in the maximum tolerated doses. With an increase

in the functional activity of the thyroid gland, small doses of thyreostatics are prescribed (thiamazole, mercazolil, 10-15 mg / day until the symptoms of hyperthyroidism are eliminated), beta-blockers (obzidan, anaprilin) at a dose of 40-80 mg / day.
Immunocorrection of autoimmune thyroiditis is carried out in accordance with the results of the immunogram: immunosuppressive therapy is prescribed with high titers of autoantibodies, that is, in the case of predominant damage to the humoral link of the immune system. To suppress the production of autoantibodies, synthetic glucocorticoids are used (prednisone at a dose of 30-40 mg / day for 2-4 weeks, dexamethasone, 3-4 mg / day in a continuous or alternating scheme every other day). Non-steroidal anti-inflammatory drugs (indomethacin, voltaren, brufen) and methylxanthine drugs (theophylline, aminophylline) have a weaker immunosuppressive effect. Indomethacin is prescribed at 25-75 mg / day from 2-3 weeks to 1 month. Theophylline is used in suppositories of 0.15 g at night for 10 days, the course of treatment is repeated up to 4 times a year with a break of 1-3 months. Immunomimetics are used for abnormalities in the cellular link of the immune system. For this purpose, thymalin is used intramuscularly: on the 1st day - 10 mg;
further - 5 mg / day up to 2 weeks; T-activin intramuscularly: 0.1% - 2.0 ml 2-3 times a week, 6-10 times. From nonspecific immunocorrectors are prescribed: dibazol, 0.006-0.004 g / day for 2 weeks (2-4 courses per year); methyluracil, pentoxil - in conventional therapeutic doses for 20-30 days (2-4 courses per year); sodium nucleate, 0.75-1.5 g / day for 10-30 days (2-4 courses per year); splenin intramuscularly, 1.0-2.0 ml for 6-10 days. When prescribing vitamins A, E, adaptogens (eleutherococcus, pantocrine, ginseng), there is also a mild immunostimulating effect on the cellular link of immunity.

GOITER ENDEMIC.

Endemic goiter is said to be when an enlargement of the thyroid gland is observed in a significant number of people living in a certain geographic area with iodine deficiency in food. It is generally accepted that the disease is endemic if 5% of children and adolescents or 30% of adults have a grade I or more thyroid enlargement.
Endemic goiter is widespread in many countries. Goiter endemic are the western regions of Ukraine, Belarus, the states of Transcaucasia and Central Asia.

Etiology and pathogenesis...

The main cause of the disease is iodine deficiency in soil, water and food. The theory of iodine deficiency was proposed in the 19th century by Prevost and Brown. This theory of geochemical iodine deficiency is still the leading one. It is confirmed by the absence of endemicity with normal iodine content in soil and water, as well as with the addition of iodine to food in conditions of endemic foci. Studies concerning the amount of iodine required by the body have shown that a

person needs 100-200 micrograms of iodine per day. With a decrease in the intake of iodine in the body to 50 μg, hypertrophy and hyperplasia of the thyroid gland occurs, that is, an endemic goiter develops. However, not all residents of the endemic area get sick. At the same time, foci of goiter endemic are known in coastal regions where there is no iodine deficiency. These facts indicate that that there must be some other goitrogenic factors. Such factors may include inappropriate nutrition with a lack of protein, ingestion of strumogenic products, unsanitary living conditions, and intoxication with chemicals.

Inappropriate nutrition, accompanied by a deficiency of protein and vitamins in food, is considered a factor provoking the development of endemic goiter. An insufficient intake of bromine, zinc, cobalt, copper, molybdenum and an excess of calcium, fluorine, chromium and manganese contribute to the development of goiter endemic. Moreover, the ratio between various trace elements in soil, water, food is important. Thus, in certain biogeochemical provinces, iodine deficiency is accompanied by a violation of the ratio of trace elements, which contributes to the development of goiter endemic. Some foods contain substances that cause the growth of the thyroid gland. Currently, the strumogenic action of thiocinates, perchlorates, thiourea, thiouracil, aniline derivatives, polyphenols has been proven. Strumogens prevent the conversion of iodides into organic iodine and thereby reduce the synthesis of iodinated hormones, which causes compensatory hyperplasia of the thyroid gland. Strumogenic substances are found in turnips, beans, rutabagas, radishes, carrots, radishes, cauliflower, peanuts, soybeans, mangoes, peaches.

Unsanitary conditions, contributing to the development of endemic goiter, lead to the appearance of protein breakdown products in drinking water - thiourea, thiouracil, urochrome, which are strumogenic.

The role of hereditary and immunological factors in the development of endemic goiter is evidenced by the fact that among the offspring of parents with goiter, this disease occurs more often than among healthy children.

Iodine entering the body is captured by the thyroid gland against the concentration gradient and serves as the main precursor for the synthesis of thyroid hormones. Iodine, entering the thyroid gland, binds to tyrosine, forming iodotyrosines, which condense into T3 and T4 molecules. The resulting hormones are found in the follicles of the thyroid gland in connection with thyroglobulin. Thyroxine is transported in a protein-bound state, cleaved off as needed and has a biological effect. Part of the thyroxine is converted to triiodothyronine.

Iodine deficiency leads to insufficient production of iodinated hormones by the thyroid gland. A decrease in the level of T3 and T4 promotes the activation of thyrotropin secretion by the pituitary gland according to the feedback law. Tyrotropin has a stimulating effect on the thyroid gland and leads to its compensatory hyperplasia and hypertrophy, which, in turn, helps to replenish the secretion of thyroid hormones. The general hyperplastic reaction can become local, as a result of which foci of medullary and cystic degeneration of the

parenchymal tissue with the development of nodular forms of goiter are formed in the thyroid gland.

Histologically, two main types of endemic goiter are distinguished: parenchymal and colloid.

Parenchymal goiter characterized by hyperplasia of the parenchyma with a microfollicular structure (i.e., many small follicles), has a tendency to form parenchymal functioning nodes.

Colloid goiter characterized by the formation of large follicles overflowing with colloid, with a flattened follicular epithelium.

The hormonal activity of such a goiter is usually reduced. Against the background of colloidal goiter, the formation of cysts, calcifications, hemorrhages is often observed.

WHO recommends the following classification of endemic goiter according to the degree of enlargement of the thyroid gland.

Group 0: goiter not.

Group I: goiter is determined only on palpation.

Group 2: goiter is determined visually in a normal position.

Group 3: goiter, detected at a distance, reaching large sizes.

According to the form of enlargement of the gland, the presence or absence of nodes, diffuse, nodular and diffuse-nodular goiter are distinguished.

Diffuse goiter characterized by a uniform increase in the thyroid gland in the absence of seals.

Nodular goiter is characterized by uneven tumor-like growth of the thyroid tissue against the background of the absence of a noticeable increase in the rest of the thyroid gland.

Diffuse-nodular goiter diagnosed in the presence of nodules against the background of a diffuse enlargement of the thyroid gland.

By localization, an atypical location of the goiter is possible: retrosternal, lingual, annular goiter.

According to the functional state of the thyroid gland, it is customary to distinguish euthyroid and hypothyroid goiter. A rare manifestation of endemic hypothyroid goiter is cretinism with impaired intelligence, growth retardation and sexual development.

Clinical picture...

The clinical manifestations of endemic goiter are determined by the degree of enlargement of the thyroid gland, its localization and functional state.

Early signs... With the development of endemic euthyroid goiter, patients complain of weakness, fatigue, headaches, and discomfort in the region of the heart.

Stage of advanced clinical symptoms...The increase in the size of the goiter leads to the appearance of signs of compression of the adjacent organs. Signs of thyroid growth can be a feeling of pressure in the neck, more pronounced when

bending over and lying down, difficulty breathing, difficulty swallowing. Compression of adjacent vessels can lead to poor circulation and expansion of the right half of the heart ("thymus heart"). The development of hypothyroid endemic goiter is accompanied by the appearance of symptoms characteristic of hypothyroidism (lethargy, apathy, lethargy, weight gain, dry skin, bradycardia, constipation, etc.). However, it should be remembered that about 50% of diagnoses of endemic goiter are euthyroid forms. Of the remaining 50% of hypothyroid forms of endemic goiter, most of them are accompanied by subclinical hypothyroidism,

FlowAn endemic goiter is determined by the degree of endemic goiter, the shape, size, localization of the goiter and the functional state of the thyroid gland. A high degree of endemic goiter leads to an increase in the incidence of endemic hypothyroid goiter. Nodular or diffuse-nodular goiter has the ability to rapidly increase in size and, more often than diffuse, causes compression of adjacent organs. The atypical location of the goiter is accompanied by the appearance of specific symptoms. Lingual goiter leads to dysphagia, choking, a change in the timbre of the voice, a feeling of tickling and sore throat. The retrosternal location of the goiter is accompanied by shortness of breath, dry cough, and the development of a “thymus heart”. Development of acute or subacute strumitis, hemorrhage in the tissue of the thyroid gland is also possible.

Diagnosis and differential diagnosis...

In the presence of an enlargement of the thyroid gland in persons living in an endemic goiter area, the diagnosis is facilitated by palpation of the neck, in which there is no increase in regional lymph nodes and a painless enlarged thyroid gland is determined. There are usually no clear clinical signs of thyroid dysfunction. Confirmation of the diagnosis is facilitated by the increased absorption of radioactive iodine by the thyroid gland. The thyroxine level corresponds to the standard values. There is a violation of the ratio of thyroxine and triiodothyronine in the blood towards the prevalence of the latter, which maintains the clinical state of euthyroidism due to the greater metabolic activity of triiodothyronine. In the process of scanning the thyroid gland, a uniform distribution of the isotope is determined in diffuse goiter and the presence of "warm" or "cold" nodes in various forms of nodular endemic goiter. With ultrasound sonography, cystic changes, areas of calcification and fibrosis, and uneven echogenicity are observed. The excretion of iodine in the urine is less than 100 μg / day.

Endemic goiterrequires differential diagnosis with Hashimoto's and Riedel's chronic thyroiditis. Common signs, ha-

characteristic of chronic thyroiditis and endemic goiter are an enlarged thyroid gland and an euthyroid or hypothyroid state in the clinical difference of autoimmune examination. Hashimoto's thyroiditis from endemic goiter consists in the presence of lymphoid infiltration during puncture biopsy of the thyroid

gland and in a high titer of antithyroid antibodies in the blood. The difference between Riedel's fibrous thyroiditis and endemic goiter is the fusion of the thyroid gland with the surrounding tissues and a very pronounced density.

Certain difficulties may arise in cases of differential diagnosis of malignant neoplasms and endemic goiter. A common symptom for these diseases may be an enlarged thyroid gland with a nodule palpable in it. The difference between a malignant neoplasm is the rapid growth of the node, its pronounced density, lack of mobility, an increase in regional lymph nodes and the presence of atypical cells in a puncture biopsy.

Treatment...

The nature of drug therapy for endemic goiter depends on the degree of enlargement of the thyroid gland and the state of its function. With goiter of the I degree, it is sufficient to prescribe potassium iodide until its size normalizes. In the course of treatment, it is necessary to remember about possible complications in the form of the Wolf-Chaikov effect, autoimmunization, and therefore it is advisable to take breaks in taking iodides during treatment or prescribe thyroid hormones containing potassium iodide - thyrocomb. When the size of the thyroid gland is 1-3 degrees, drug therapy is carried out with synthetic thyroid hormones in doses: thyrotome - 40 - 80 mcg / day, levothyroxine or eutirox - 100 mcg / day. If symptoms of an overdose appear, the dose is reduced or the drug is canceled for 2-3 weeks.

Indications for surgical treatment are determined by the presence of nodes, especially "cold" ones that do not absorb isotopes, the rapid growth of the goiter, the presence of signs of compression of the surrounding organs and tissues, and suspicion of malignancy. After the operation, it is advisable to prescribe thyroid hormones to prevent goiter recurrence.

Prophylaxis endemic goiter is divided into mass, group and individual.

1. Mass preventioncarried out by iodization of table salt by adding potassium iodide at the rate of 25-40 g per 1 ton. The quality of iodization is controlled by the sanitary-epidemiological service. It should be remembered that the iodide content in table salt is influenced by the conditions of its storage. Destruction occurs at high humidity. In addition, iodized salt should be added to food after it has been prepared, since high temperatures also inactivate iodine contained in salt. One of the components of mass prevention is a sufficient protein content in food (not less than 1.0 g per 1 kg of body weight).

2. Group preventioncarried out in children and pregnant women, taking into account their increased need for thyroid hormones. Children from 4 to 10 years old are prescribed potassium iodide based on the daily requirement (25 μg / day) in addition to the diet with iodine-containing foods; from 10 to 17 years old - 50 mcg / day, for pregnant women - 50-75 mcg / day. It must be remembered that potassium iodide is indicated for group prophylaxis for people who do not have goiter, but who have an increased need for thyroid hormones. The control over

the sufficiency of the dose is the excretion of iodine in the urine. Prolonged drugs containing potassium iodide (lipoidol) are prescribed one capsule 1-2 times a year.

3. Individual preventionappoint underwent strumectomy, temporarily residing in endemic areas, exposed to strumogenic factors in everyday life or at work. It is carried out by the inclusion in the diet of foods rich in iodine (seaweed, squid, sea fish, persimmons, walnuts) or the intake of potassium iodide.

All types of iodine prophylaxis do not eliminate strumogenic environmental factors. In endemic areas, they are aimed only at providing the body with iodine - one of the most important components for the biosynthesis of thyroid hormones. That is why the cessation of goiter prophylaxis naturally leads to an increase in the incidence.

THYROTOXIC ADENOMA

Thyrotoxic adenoma - a disease characterized by the presence of a node (adenoma), autonomously increased producing thyroid hormones, hypoplasia and decreased function of the rest of the thyroid gland.

Thyrotoxic adenoma, as a rule, is observed in women, more often over the age of 40.

Etiology and pathogenesis... The etiology of thyrotoxic adenoma, like other adenomas, is not clear enough. Thyrotoxic adenoma produces thyroid hormones autonomously, regardless of the action of pituitary thyroid stimulating hormone. As a result of increased production of thyroid hormones, suppression of TSH production is possible, followed by a decrease in the function of the rest of the thyroid gland.

Clinical picture... The clinical picture of thyrotoxic adenoma is basically the same as in diffuse toxic goiter. Unlike the latter, thyrotoxic adenoma often has a low-symptom course with a moderately pronounced clinical picture (slight weakness, slight weight loss, moderate tachycardia, atrial fibrillation), the absence of endocrine ophthalmopathy and pretibial myxedema. On palpation of the thyroid gland, an elastic node is determined, with clear boundaries and a smooth surface of varying density, freely moving when swallowing. Thyroid-stimulating antibodies are not detected in the blood.

Diagnostic tests... Tests with triiodothyronine (thyroxine) and with thyroliberin are negative, which confirms the autonomy of a functioning thyroid adenoma. The scan shows a high uptake of radioactive iodine by the node ("hot" node) with reduced uptake in the rest of the gland.

Diagnosis and differential diagnosis.

Diagnosis based on the analysis of clinical signs (weight loss with good appetite, emotional lability, stable tachycardia, increased pulse blood pressure, thyroid

hyperplasia, eye symptoms, tremors of the hands and the whole body). Difficulties in making a diagnosis may be associated with the multisymptomatic course of the disease or the predominance of one of the symptom complexes. As a rule, with a detailed clinical picture of thyrotoxic adenoma, the diagnosis is not difficult. In the case of initial and mild forms of this disease, the following criteria are of diagnostic value:

- an increase in the content of T4 and T3 in the blood serum (T4 more than 140 nmol / l, T3 more than 2.0 nmol / l);
- an increase in the level of thyroglobulin in the blood serum, a decrease in the level of TSH less than 0.6 mIU / l;
- on the ECG: sinus tachycardia, sinus arrhythmia, high voltage of the teeth, atrial fibrillation, biphasic or negative T wave;
- shortening of the Achilles reflex time less than 180 m / s (230-270 m / s);
- in the biochemical analysis of blood - hypocholesterolemia, hypoalbuminemia, there may be hyperbilirubinemia, an increase in the level of transaminases;
- in the general analysis of blood there may be leukopenia, absolute or relative lymphocytosis, relative or absolute neutropenia, thrombocytopenia, anemia;
- changes in the immunogram: on the part of the humoral link of immunity - the presence of thyroid-stimulating antibodies in the blood serum, antibodies to thyroglobulin;
- on the part of the cellular link of immunity: a decrease in the number of total T-lymphocytes, a decrease in T-suppressors, an increase in the number of activated T-lymphocytes,
- on ultrasound, there is an increase in the size of the thyroid gland, uneven changes in echogenicity with foci of its decrease.

Depending on the leading clinical syndrome, thyrotoxic adenoma is necessary ***differentiate*** from the following diseases:

1. Neurocirculatory dystonia and climacteric neurosis.
2. Rheumatic heart disease and rheumatic heart disease.
3. Atherosclerotic cardiosclerosis and aortic atherosclerosis.
4. Chronic enterocolitis.
5. Tuberculosis.
6. Malignant neoplasm of the thyroid gland.

Common signs of neurocirculatory dystonia and thyrotoxic adenoma there may be emotional lability, sweating, tachycardia, a tendency to arterial hypertension.

The difference thyrotoxic adenoma consists in progressive weight loss, stable tachycardia, even at rest, during sleep. An increase in pulse pressure, the presence of eye symptoms and an enlargement of the thyroid gland, along with indicators of additional diagnostic methods (high levels of T4, T3, low plasma cholesterol).

General clinical symptoms of climacteric neurosis and thyrotoxic adenoma are: irritability, poor sleep, tearfulness, feeling of heat, increased sweating.

The difference Thyrotoxic adenoma consists in increasing weakness and weight loss, while in the climacteric period there is a tendency to increase body weight, in addition, the feeling of heat in patients with thyrotoxic adenoma is constant, and in the climacteric period it is noted as "hot flashes", replaced by a feeling of chilliness. Additional research methods help formulate the correct diagnosis.

Common signs of rheumatic heart disease and thyrotoxic adenoma*:* low-grade body temperature, palpitations, shortness of breath, general weakness, pain in the heart.

The difference thyrotoxic adenoma in the stability of tachycardia, sonorous, increased heart sounds, progressive weight loss with good appetite, as well as in the presence of eye symptoms and an enlarged thyroid gland.

Differential diagnosis with aortic atherosclerosis carried out according to the general symptom of an increase in pulse pressure in cases of mild clinical signs of thyrotoxicosis in elderly and senile people.

The difference thyrotoxic adenoma consists in the stability of tachycardia, which is not amenable to treatment with glycosides, emotional lability, a tendency to sweating, in a decrease in blood cholesterol and an increase in the content of thyroid hormones.

Treatment...

Treatment of thyrotoxic adenoma is most often surgical - subtotal resection of the affected lobe of the thyroid gland with adenoma. Surgical treatment is carried out only when the euthyroid state is reached by means of preoperative preparation.

In case of contraindications to surgery, treatment with radioactive iodine can be used.

Attachment 1

DIAGNOSTIC SIGNIFICANCE OF HORMONAL PARAMETERS,

Tz in the blood:

increased: thyrotoxicosis, early failure of the thyroid gland, with iodine deficiency;

downgraded: hypothyroidism.

Total PM in blood:

promoted: thyrotoxicosis, conditions with an increased level of thyroxin-binding globulin (pregnancy, genetically determined disorders), acute thyroiditis;

downgraded: hypothyroidism, a condition with a low level of thyroxine-binding globulin (genetically determined, nephrotic syndrome, chronic liver disease), panhypopituitarism.

Free PM in blood:
increased: thyrotoxicosis,
downgraded: hypothyroidism.

TSH in the blood:
increased: primary hypothyroidism, autoimmune thyroiditis with clinically pronounced hypothyroidism, ectopic TSH secretion (lung and breast tumors), subacute thyroiditis,
downgraded: secondary hypothyroidism, thyrotoxicosis.

CHANGE OF THYROID HORMONES T TSH IN DISORDERS OF THE HYPOTHALAMO-HYPOPHYSICAL-THYROID SYSTEM.

Pathology	Description	T4, T3	TSH
Primary hypothyroidism	Insufficiency of the thyroid glands	Downgraded	Promoted
Secondary hypothyroidism	Dysfunction of the pituitary gland	Downgraded	Normal or downgraded
Tertiary hypothyroidism	Hypothalamic dysfunction	Normal or downgraded	Normal or downgraded
Hyperthyroidism	Graves' disease, functional thyroid autonomy	Promoted	Downgraded

NOTES

1. TSH is characterized by a diurnal rhythm: the highest blood level is observed in the morning at 6 o'clock, the minimum values - in the evening at 17-18 o'clock. Laboratory research is carried out in the morning on an empty stomach.
   The determination of TSH is influenced by high levels of lipidemia.
   Normally, the level of TSH in the blood of newborns is significantly increased at birth, significantly decreases by the end of 1 week of life, and finally normalizes by the end of 1 month. The optimal timing of examination of newborns in order to identify hypothyroidism is the 5th day from the moment of birth. If the TSH level exceeds 20 mIU / L (threshold), a new blood sample must be retested. Concentrations of the order of 100 mIU / L and higher are typical for congenital hypothyroidism.
   In adults, the concentration of TSH is not subject to strong fluctuations.

The lower limit of normal values for TSH is not clearly defined. Some individuals may have low TSH values without clinically evident symptoms of hyperthyroidism.

2. The maximum concentration of the hormone T4 total (T4) and free T4 (T4F) is determined from 8-12 hours, the minimum - from 23 to 3 hours. During the year, the maximum values of hormones are observed between September and February, the minimum - in the summer.
   During pregnancy, the concentration of T4 increases, reaching maximum values in the III trimester, which is associated with an increase in the content of thyroxine-binding globulin under the influence of estrogens. In this case, the content of T4F may decrease.
   In severe diseases not associated with the thyroid gland, the concentration of T4F, as a rule, remains within the normal range, and T4 may be reduced.
   At the initial stage of hypothyroidism, the T4F level decreases earlier than the T4 level.

3. Seasonal fluctuations are typical for T3 and T3F: the maximum level is from September to February, the minimum is the summer period.
   By the age of 11-15, the concentration of the hormone reaches the level of adults.
   Determination of T3F serves mainly to confirm the hyperthyroid status and is of very limited value in the diagnosis of hypothyroidism.
   During pregnancy, T3F decreases from 1 to III trimester, a week after childbirth - it normalizes.
   Elderly people, as well as patients suffering from severe general somatic diseases, often have a so-called low T3 syndrome with a normal T4 content. Low T3 syndrome in this subgroup of patients, which develops due to an increase in the level of reverse T3, is not a sign of hypothyroidism.

4. The level of antibodies to thyroglobulin and to thyroid peroxidase is used only in combination with the results of other tests and the clinical manifestations of the patient.
   At low concentrations, autoantibodies can be found in 10% of the healthy population and in patients with diseases not associated with the thyroid gland, for example, in inflammatory rheumatic diseases.

Appendix 2.

RECIPES

Rp. Tab. "Antistruminum" N. 30
DS 1 tablet once a week

Rp. Iodi Natrii jodidi 2.0
Aq. Destil. ad. 100 ml steril.
VDS 100-50 drops in 1 liter of 5% glucose solution
on isotonic sodium chloride solution drip
intravenously

Rp. Tab. Mercasolili 0.005 N. 20
DS 2 tablets 3 times daily with meals

Rp. Tab. "Microiodi" abductae N. 40
DS 1 tablet 3 times a day after meals in courses
20 days each with 10-20 day breaks

Rp. Sol. Natrii iodidi 10% 10 ml
DS Inject 5-10 ml intravenously every 8 hours
(with thyrotoxic crisis)

Rp. Parathyreoidini 1 ml
DtdN 6 in ampull.
S. 1 ml 2 times a day intramuscularly

Rp. Tab. Thyreocombi N. 40
DS 1 tablet 1 time in the morning before breakfast

Rp. Tab. Triiodthyronini 0.00002 N. 50

DS 1 tablet 1 time daily in the morning before meals

Rp. Tab. L-thyrocsini 100 N. 50

DS 1 tablet in the morning before meals

Rp. Tab. Euthyrocsidi 25 N. 50

DS 1 tablet in the morning before meals