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Кафедра Внутрішньої медицини №3
Факультет VI по підготовці іноземних студентів

ЗАТВЕРДЖЕНО
на засіданні кафедри внутрішньої медицини №3
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Зав. кафедри _____д.мед.н., професор Л.В. Журавльова

МЕТОДИЧНІ ВКАЗІВКИ
для студентів

з дисципліни «Внутрішня медицина (в тому числі з ендокринологією)
студенти 4 курсу I, II, III медичних факультетів, V та VI факультетів по підготовці іноземних студентів

Йододефіцитні захворювання щитоподібної залози. Ознаки ендемічної місцевості за ВООЗ. Клініка, діагностика, профілактика та лікування. Гіпотиреоз та тиреоїдити. Класифікація, діагностика, клініка, лікування.

Харків 2016

Topic- «Iodine deficiency diseases of thyreoid gland. Clinic, diagnostic, prophylaxis and treatment. Hypothyroidism and thyroiditis. Classification, diagnostics, clinic, treatment»

1.The number of hours – 5

Practical tasks of lesson:

1. To teach the method of determination of etiologic and pathogenic factors and treatment of iodine deficiency.
2. To teach the method of determination of etiologic and pathogenic factors of hypothyreosis (HT) and thyroiditis (T).
3. To acquaint students with classifications of HT and T
4. Determination of typical clinical picture of HT.
5. An acquaintance is with the clinical variants of T.
6. An acquaintance of students is with possible complications of HT.
7. Working off methodology of determination of basic diagnostic criteria of HT.
8. Working off methodology of determination of basic diagnostic criteria of T.
9. Drafting of plan of inspection of patients is on HT.
10. Drafting of plan of inspection of patients is on T.
11. Analysis of results of laboratory and instrumental researches which are used for diagnostics of HT.
12. Analysis of results of laboratory and instrumental researches which are used for diagnostics of T.
13. Tactic of conducting of differential diagnostics of HT.
14. Tactic of conducting of differential diagnostics of acute T.
15. Tactic of conducting of differential diagnostics of chronic T.
16. Technology of ground and formulation of diagnosis of HT .
17. Drafting of plan of treatment of patients is on HT and T.

What must a student know?

1. Determination of concept of HT, T.
2. Epidemiology of HT and T in Ukraine.
3. Factors of risk of HT and T.
4. A mechanism of hormonal and metabolic violations at HT and T.
5. Etiology and pathogenesis of HT and T.
6. Clinical picture of HT and T.
7. Typical clinical picture of HT.
8. Polyorganic complications of HT.
9. Diagnostic criteria of HT and T.
10. Testimony to conducting and analysis of results of hormonal researches and tests.
11. Diagnostic value of ultrasonic research of thyroid, radioisotope research of thyroid (radiometry, scanings).
12. Choice of method of treatment of HT and T.

What must a student be able?

1. To diagnose iodine deficiency.
2. To define the factors of risk of HT and T.
3. To diagnose HT and T.

4. To carry out palpation research of thyroid.
5. To determine the degree of multiplying a thyroid.
6. To diagnose the syndrome of HT.
7. To determine grade of syndrome of HT.
8. To define character of polyorganic complications of HT.
9. To analyze the results of hormonal researches and functional tests.
10. To estimate the results of ultrasonic and radioisotope research of thyroid.
11. To conduct differential diagnostics of syndromes of HT.
12. To estimate dynamics of thyroidal status of patients on a background the use of thyroidal preparations.
13. Able to correct the dose of thyroidal preparations and concomitant preparations on the measure of achieving the thyroidal state for patients.
14. Drafting of long-term plan of HT, prophylaxises and treatments of his complications, seasonal correction of doses of substitute therapy; technology of bringing in of patient is to participation in a medical process.
15. Co-operating with contiguous specialists (by a surgeon, oculist, cardiologist, neurologist) on the stage of establishment of complete diagnosis and protracted supervision of patient on HT or T.

Content of the topic <u>Definition of iodine deficiency. "Manifestations of iodine deficiency</u>.

Local symptoms are associated directly with a goiter as such. Typical complaints: feeling of pressure in the neck, sudden attack of coughing through the stimulation of recurrent nerve.

Insanity and headaches are caused by compression of large vessels in the neck area. Because of compression of large vessels may be a swelling of the face.

Circulatory disturbance spread to lesser circulation and leads to hypertrophy and dilatation of the right ventricle, develops so-called «goitre heart».

As a result of the pressure of goiter on the trachea breath rises, asthma attacks may occur. Sometimes there is discomfort in swallowing as a result of compression of the esophagus. It turns out a number of vegetative disorders associated with stimulation or inhibition of the sympathetic nerve trunk and other nerve structures.

With the development of hypothyroidism that accompanies expressed endemic goiter, there is a characteristic clinical picture. Hyperthyroid form of the disease is usually run lightly than the primary thyrotoxicosis. As a result of endemic goiter lesion of few generations endemic cretinism appears.

<u>Determination of iodine-deficiency areas for goiter prevalence in different age groups and data urinary iodine.</u>

Iodine deficiency leads to insufficient production of thyroid hormones and decrease of their secretion. By principle of feedback reduce of level of thyroxine in the blood causes the stimulation of thyrotropin. This stimulation is carried both humoral and neuro-reflexive was in the form of impulses of the receptors of nerve endings in the thyroid gland. Increased level of thyrotropin causes compensatory hyperplasia in thyroid tissue, which helps improve hormonopoiesis in thyroid gland and ensuring adequate level of thyroxine.

In the development of compensatory reaction involved not only thyrotropin, but also hypothalamic releasing hormone - tiroliberin. In some cases the process is limited by compensatory hyperplasia of the thyroid gland, which fills the deficiency of thyroid hormones in conditions of exogenous iodine deficiency. Another compensatory mechanism is increasing of synthesis of triiodothyronine, which has a greater hormonal activity.

Endemic goiter distributed in the mountains and some plains regions of Ukraine (Carpathians). Prevalence of endemic goiter is determined by a number of indicators: 1) the correlation of men and women with goiter (an indicator of the Lenz-Bauer, than it is closer to unity, the harder it is endemic), 2) the prevalence of nodular forms of goiter 3) the presence of cretinism; 4) animals with goiter, 5) the number of people with thyroid hyperplasia.

Endemic is difficult, if the frequency of lesions of the population above 60%, the index of the Lenz-Bauer 1/3-1/1, and the frequency of nodular goiters is higher than 15%, there are cases of cretinism.

In mild endemic frequency of lesions of the population above 10%, the index of the Lenz-Bauer 1 / 6, the nodal forms are found in 5% of cases.

Indicators of iodine metabolism

Butanol extractable iodine (BEI) in the blood consists of thyroxine and a small amount triiodothyronine. Protein-bound iodine (PBI) in the blood consists of thyroxine (90-95%) and mono- and diiodthyrozine.

Indicators of PBI more 670 nmol / L and BAY greater than 440 nmol / L indicate hyperthyroidism.

These two methods are very demanding, especially the definition of BEI, and inaccurate. The study of absorption of labeled triiodothyronine by erythrocytes used in patients who did not receive antithyroid drug therapy, and make out 11%. It increases depending on the severity of the disease.

Studies are uninformative in the cases when patients used iodine-containing medications or in cases of concomitant reduction of plasma proteins, reduction of the level of thyroglobulin.

Iodine prophylaxis: mass, group, individual. The value of consumption of iodized salt in prevention of iodine deficiency disorders. Restriction for the use of drugs based on potassium iodide.

The most convenient method of ***mass iodine prophylaxis*** is the use of iodized salt. Iodized salt contains 25 grams of potassium iodide in 1 tone of salt. Together with iodine prophylaxis in endemic areas should begin to romp about salt-water fish and other seafood.

Group iodine prophylaxis is conducted by antistrumin (1 pill contains 0,001 g of potassium iodide) in organized groups of children, to pregnant and lactating women.

Individual iodine prophylaxis is carried out to persons who had undergone surgery for endemic goitre, persons who are temporarily residing in areas of endemic goiter.

Determination of size of thyroid gland.

Thyroid gland normaly is invisible and is not defined during palpation. During palpation we are looking for the thyroid gland in the front of the neck, near the lower edge of thyroid cartilage on either sides of him. The thyroid gland gives the impression of roller, which rolls during the swallowing movements.

On the view we should pay attention to the shape of the neck, the presence of pulsations and outpouching of thyroid gland. Most thyroid diseases are accompanied by increasing of thyroid gland, but absence of increased thyroid gland does not exclude the presence of pathology. The thyroid gland may be enlarged diffusely or through its individual parts.

During palpation pay attention not only on quantity, but also at the location of the thyroid gland and its texture (elastic, dense, soft, and woody). Assess the nature of the surface of the thyroid gland (faty, bumpy), mobility, presence of pain during palpation. Thyroid enlargement is observed in diffuse toxic goiter, sporadic and endemic goiter, inflammatory diseases, and cancer.

Definition - "goiter. "

The clinical term ***"nodular goiter"*** combines focal thyroid lesions, which have different pathmorphological structure - the cyst, these nodes, benign and malignant tumors of the thyroid gland. Tumors of the thyroid gland in most cases are of epithelial origin - an adenoma and adenocarcinoma.

Endemic goiter – is a disease that occurs in certain biogeochemical geographic areas with iodine deficiency in the environment, which is characterized by enlargement of the thyroid gland.

Sporadic goiter is persistent increase of thyroid gland among residents of areas without iodine deficiency (a physiological providing of iodine).

Goiter is enlargement of the thyroid gland third degree or higher. The increase thyroid gland 2 nd degree is called thyroid hyperplasia, but in the presence of node this variant is also called goiter.

WHO classification developed to work in areas with endemic goiter:

Group 0. No goiter.

Group 1. Goitre determined by palpation. The thyroid gland is clearly visible when head is thrown back and neck is elongated.

Group 2. Goiter is determined visually.

Group 3. Goitre is seen at a distance, grows up to large sizes, mechanicaly makes breathing difficult.

By the form of increased thyroid gland, the presence or absence of nodes are distinguished:

- Nodular goiter (characterized by tumorlike growth of thyroid tissue often round in shape, mainly it has elastic consistency, other sections of thyroid gland usually do not palpate);

- Diffuse goiter (characterized by an even increase of the thyroid gland in the absence of local seals);

- Mixed, or diffuse nodular goiter (union of the diffuse hyperplasia and node).

Hypothyreosis, etiology, pathogenesis and clinical signs.

Hypothyrosis is a syndrome which develops as a result of sharp decline of function of thyroidal gland.

Term "myxedema" is traditionally used for description of the most heavy forms of hypothyrosis, means the mucus edema of skin and hypodermic cellulose.

Hypothyrosis - comparatively rare disease which meets mainly for women 30-60 years, although some growth of number of patients is the last years marked with the different forms of hypothyreosis.

Aetiology

In 95% patients on hypothyreosis it is primary. He can be born or purchased. Born hypothyreosis develops at aplasia or hypoplasia of thyroidal gland as a result of violations of pre-natal development or as a result of genetic defects of synthesis of thyroidal hormones.

The primary is purchased hypothyreosis arises up for a number of reasons.

1. On this time hypothyreosis is most widespread, that arises up as a result of chronic autoimmune thyroiditis . Thus as a result of growth of title of circulating autoantibodies to tissue of thyroidal gland there is a reaction with cellular antigens with subsequent development of destructive and proliferative processes. In the total there is a decline of products of thyroidal hormones.

2. As complication of medical measures after:

а) operative treatment (subtotal or total resection of thyroidal gland) - makes the third part of all hypothyreosis;

б) treatment of diffuse toxic goitre by a radio-active iodine;

в) badly controlled inadequate or protracted treatment of thyreostatic preparations (merkazolilum, preparations of lithium);

г) use of surplus doses of iodine preparations;

д) radial therapy of malignant new formations of organs which are located on a neck;

е) protracted reception of hormonal preparations (glucocorticoids, sexual hormones) or sulphanilamide.

3. Destructive defeats of thyroidal gland: malignant tumors, acute and chronic infections (thyroiditis, abscess, tuberculosis, toxoplasmosis).

4. As a result of the insufficient entering organism of iodine.

Pathogenesis

At hypothyreosis all types of exchanges are repressed та utilization of oxygen by tissues, oxidizing reactions are braked and activity of the different enzymic systems, interchange of gases and basic exchange, goes down, thermoregulation is violated. There are violations of albuminous exchange, which result in deceleration of disintegration and synthesis of albumen, violation of exchange of glucose-aminoglycans, accumulation in tissues of glucoproteide mucin, gialuronic acid. Their surplus changes the colloid structure of connecting fabric, multiplies its hydrophylity and sodium, that in the conditions of myxedema. Surplus of vasopressine can also influence on the mechanism of delay in tissues of water and sodium, the products of which are braked thyroidal hormones.

Violation of lipid exchange appears the declines of synthesis and disintegration of lipids. Maintenance of cholesterol rises, threeglycerids, β-lipoproteids (II and IV type of hyperlipoproteidaemia).

A carbohydrate exchange is violated - suction of glucose in an intestine and its utilization is slowed in an organism.

The deficit of thyroidal hormones brakes development of tissues of brain and represses higher nervous activity. Hypothyroidal encephalopathy which an intellect and psychical activity goes down at develops, conditional and absolute reflex activity grows weak.

Activity of other ductless glands goes down (in particular, adrenal cortex in the conditions of hypothermia). Peripheral metabolism of hormones of endocrine glands is violated (corticosteroids and sexual hormones).

Clinical displays

A disease develops slowly, patients remember the first signs of illness heavily, besides initial displays are characterized wretched and unspecific symptomatic, that is why patients can lasted and unsuccessfully to treat oneself concerning the different diseases of the cardiac or nervous system.

Speed of development and expressed of symptoms of hypothyreosis depend on reason of disease, degree of thyroidal insufficiency and individual features of patient.

1. Thyroidal gland

At palpation the state of gland can be different, in dependence on a disease which entailed the origin of hypothyrosis. At primary hypothyreosis the thyroidal gland is not access for palpation quite often, the presence of goitre is although possible. At second hypothyreosis the thyroidal gland is more frequent megascopic.

2. Change of the state of skin and hypodermic cellulose

At expressed hypothyreosis patients are very alike on each other: pale, puffy, mask-face with narrow eye cracks, lines of face are crude, large, thickened.

A skin is pale, with a wax or marble tint through decelerations of peripheral blood stream and anaemias, which accompany a syndrome often. Sometimes there is a bright blush of cheeks. The icteric of skin is possible, especially on hands, as a result of surplus of β-carotin which is slowly transformed in a liver by the vitamin A.

A skin is cold, the temperature of body for patients on hypothyreosis is reduced. Infectious diseases and inflammatory processes can for them develop without the expressed temperature reaction. These violations are the intensities of power exchange related to the decline and by the delay of liquid in an organism. Patients constantly are warmly dressed (at any time to the year), as a decline of basic exchange and violation of thermoregulation diminish tolerance to the cold.

A skin is dry, peels, with the areas of cornification. In connection with an accumulation a glucosaminoglycans the skin becomes dense, thickened, not going to the folds.

A symptom of Bar is a surplus cornification and bulge of epidermis on knees and elbows, sometimes on a rear feet and internal stone. Hereupon a skin in these areas becomes dirty quickly (symptom of dirty knees and elbows).

Syndrome of Villanova-Kanyadel - hypothyroid dermopathy: dry skin with promoted follicle hyperkeratosis mainly on the external surface of shoulders and thighs, on the back, insignificant general hyperpigmentation.

Typical edema of extremities, especially brushes and feet. Extremities are thickened, the fingers of hands are thick and make impression of short. Supraclaviculary pits are filled. Actual hydrostatical edemata are possible on shins, trunk as a result of delay of destroying of sodium.

Hairs on a head are dry, fragile, liquid. The fall of cilia, hairs appears in the area of external third of eyebrows, in axeles, pubes.

A symptom of Rotschild is fragility nails, fragility and fall of hairs, especially on external third of eyebrows.

Nails, scratched all over, grow slowly.

Quite often there is ptosis, anomalies of refraction. Ophthalmopathy is rare, without a tendency to progress.

3. Hypothyreoid myopathy

Shows up by pseudohypertrophy of muscles, myasthenic and myotonic syndrome. The expressed of myopathy correlates with weight of hypothyrosis. Pseudohypertrophy of muscles is characterized multiplying muscular mass, underlined relief of muscles. Muscles are dense.

The myasthenic syndrome is characterized the decline of muscular force, increase of muscular fatigueability, appearance of muscular pain (myalgias), especially in the proximal group of muscles. Muscular pains are accompanied a myotonic syndrome - cramps, deceleration of relaxation of muscles.

A syndrome of Hoffmann is combination of muscular hypertrophy, sickly muscular spasms and pseudomyotony.

4. Defeat of the peripheral nervous system - polyneuropathy

Violation of function of cranial nerves, motive and sensible disorders, neuralgias, appearance of pathological reflexes, is possible, anisoreflexia

The changes of the peripheral nervous system appear pains in extremities, by cramps and flow asradicukirtis or polyneuritis.

5. Psychoemotional violations

For patients on hypothyrosis characteristic changes of the central nervous system or other degree of psychical disorders is observed for all patients, and sometimes they prevail in clinical symptomatic.

Characteristic languor, promoted fatigueability, decline of capacity. Violations appear in a motivational sphere is indifference, absence of interest to everything, that surrounds. Together with psychical indifference there can be the promoted crabbiness, nervousness, officiousness. Psychical reactions are slow on external irritants, speed of motive reactions is reduced.

Memory and intellectual capabilities gets worse. Patients can not attract attention. Quite often patients sick can not execute work, related to the intellectual loadings. The typical twisting of formula of sleep is a somnolence in the day-time, insomnia at night. Often there is persistent head pain, dizziness, noise in ears.

At expressed hypothyrosis heavy chronic hypothyroid developspsychosyndrome, that acquires the lines of psychoses, manic-depressive syndrome.

An ear is reduced as a result of edema of mucus shell of middle ear.

6. Defeat of the cardiac system

"Hypothyroid heart"

At hypothyrosis the cardio-vascular system suffers considerably. The defeat of myocardium with subsequent development of "hypothyroid" heart shows up already on the early stages of disease. Violation of processes of exchanges lies in basis of changes, peculiar hypothyrosis, oppressing oxidizing processes, insufficiency of coronal circulation of blood. Specific changes in myocardium (it was swollen, swelling, muscular degeneration) loosen his retractive ability, causing diminishing of shock volume and cardiac troop landings, decline of volume of циркулюючої blood and lengthening time of circulation.

The shortness of breath arises up at the minimum physical loading.

Pains in the area of heart, retropectoral, unconnected with the physical loading, as a rule, not removed adopting nitroglycerine.

Mechanism of cardialgia: 1) coronagenic, 2) metabolic. The high doses of thyroid hormones multiply utilization of oxygen sharply, promote the necessity of myocardium in oxygen, can provoke appearance of sthenocardia or development of cardiac insufficiency.

There is tonogenic dilatation of cavities of heart, multiplying the sizes of heart with expansion of his scopes, loosening a cardiac pulsation.

Tones of heart are muffled auscultative.

Bradicardia appears in most patients (30-60%), the amount of cardiac reductions is sometimes diminished to 40/min.

But bradicardia can be not in a number of cases, or she changes on tachycardia (in 10% patients) at presence of the expressed anaemia or cardiac insufficiency.

Violations of rhythm are very rare.

A pulse is small, soft.

Arterial pressure is reduced or normal, in 10-50% patients is high blood pressure. Factors which are instrumental in the increase of level of arterial pressure is multiplying peripheral resistance of arteries is promoted. It is impossible to eliminate the role of hormonal factors (increase of excretion of вазопресину, increase of concentration of noradrenalin at the normal level of adrenalin, change of activity of renin in plasma). Arterial hypertension can diminish and even disappear under act of adequate thyroid therapy.

For patients on hypothyrosis, especially sear and yellow leaf, often as a concomitant disease develops ischemic heart trouble, atherosclerosis, hypertensive illness, insufficiency of circulation of blood.

Hydropericardium

The accumulation of liquid in a pericardium is observed in 30-80% patients. A liquid accumulates gradually, slowly and can achieve a volume from 15-20 to 100-150 ml, however tamponade hearts at hypothyrosis meets very much rarely. It confirms circumstance that adequate treatment allows to decrease the amount of liquid in a pericardium. Sometimes hydropericardium is an unique symptom of hypothyrosis. At expressed polyserositis other symptomatic of hypothyrosis it can be not enough expressed.

Pericarditis can be combined with other displays of hypothyroid polyserositis –hydrothorax, mainly autoimmune genesis.

7. Defeat of bones

Defeats of bones, as a rule, untypical, determines only at the protracted and heavy motion. Moderate osteoporosis, conditioned the decline of maintenance of mineral matters and insufficient synthesis of albumens can develop.

Not uncommon arthralgia, arthropathia, arthrosis. Permanent pains in small of the back, which are removed during indemnification of thyroid exchange, are possible.

The defects of epiphysary ossification must put with uncompensated hypothyrosis, bone age falls behind from chronologic, linear growth is slow, extremities are shortened.

8. Defeat of digestive system

A tongue is megascopic in a volume, on the lateral surface of dent from teeth, assessed a greyish raid, feelings of tastes and appetite are reduced. As a result of multiplying the sizes of tongue the episodes of apnoe are possible in sleep, an articulation - language slow, unclear is broken.

Secretory and the excretory functions of stomach are reduced, that is accompanied the decline of appetite, nausea, pains are possible in the overhead half of stomach, vomit.

Motoric of intestine which results in development of atonic constipations is slow, sometimes there is a clinical picture of dynamic impassability of intestine. The suction function of intestine which results in proof flatulence is reduced.

The detoxic and synthetic function of liver is reduced. Quite often there is dyskinesia of bilious ways for hypomotoric type. The decline of tone and motoric of bile-excretion ways conduces to violation the bile-excretion function of liver, stagnation of bile, assists development of bile-stone illness.

9. Defeat of kidneys

The functions of kidneys and urine ways change unimportant. However much a kidney blood stream goes down as a result of violation of peripheral haemodynamic, violation of balance of

vasopressine, filtration which results in the decline of destroying of urine kidneys diminishes, to the delay of sodium and water in an organism.

Easy proteinuria is possible.

The atony of urine ways assists development of urogenital infections.

10. Disfunction of breathing organs

Violation from the side of organs of breathing expressed unimportant. Through swelling of mucus shell of nose can be difficult nasal breathing, excretions appear from a nose. The phenomena of vasomotoric rhynitis, caused by hypothyrosis, are observed during throughout the year, but more expressed in dry weather.

It was swollen and bulge of vocal connection is accompanied the change of timbre of voice - to appearance of deeper, rough, hoarse voice.

Typical edema of mucus shell of respiratory tracts.

As a result of dyscoordination of muscular reductions, violation of the central adjusting observed alveolar hypoventilation of lungs with hypoxy. Vital capacity of lungs something reduced through the weakness of intercostal muscles or oppressing a respiratory center.

Patients are inclined to the diseases of respirators - bronchitis, pneumonias, as a rule, with the languid protracted motion, without the expressed temperature reaction.

11. Thyrogenic anaemia

Anemia is conditioned achloridehydria, by the reduced suction of Fe, vitamins of B12, PP, oppressing the processes of exchanges in marrow. Anemia are possible autoimmune genesis. On occasion it is found out anemia to appearance of clinical signs of hypothyrosis.

Anemia can be normo-, hypo- and hyperchromic.

12. Endocrine violations

Considerable changes are determined from the side of the endocrine system.

The function of adrenal cortex goes down, especially at heavy decompensated hypothyrosis.

Women have violation of ovarial-menstrual cycle. A capacity for conception is saved, but there can be fruitlessness. Not uncommon complications of pregnancy are gestosis, abortions on different terms, premature births.

A libido and potency goes down for men.

Investigations.

Haemogramm. Change of indexes of clinical blood test appear at more greater part of patients. There are normochromic, normo- or macrocytary anemia with insignificant anisocytosis.

The number of leucocytes does not change, relative lymphocytosis is determined.

Biochemical violations. At hypothyrosis violated albumen, carbohydrate, fatty exchanges.

Violation of albuminous exchange. The synthesis of albumen and disintegration at hypothyrosis is reduced, that is why in blood, diminishing of maintenance of general albumen is determined and dysproteinaemia due to multiplying maintenance of globulins of gamut.

Violation of carbohydrate exchange. Sugar of blood on an empty stomach more frequent normal, sometimes reduced as a result of deceleration of suction of glucose in an intestine.

Hypothyrosis is rarely combined with saccharine diabetes - at decompensated hypothyrosis necessity goes down in insulin.

Violation of lipid exchange. Characteristic increase of synthesis of cholesterol and decline of his catabolism- the level of cholesterol rises (to 12-14 mmol/l).

Maintenance of general lipids is promoted, general three-glycerids and atherogenic factions of lipoproteins- normo of low closeness and normo of very low closeness – pre-β- and β-proteins.

The concentration of non-aetherificated fat acids is reduced.

At the same time for some patients a lipid spectrum substantially is not violated.

Violation of iodine balance

Functional insufficiency of thyroidal gland is characterized the decline of accumulation of iodine, related to the proteins of blood.

Radiometry of thyroidal gland

Hypothyrosis is accompanied the decline of absorption of I^{131} by a thyroidal gland, mainly in 24-72 hours (at a norm 25-50% indexes do not exceed 10-15%). However it should be noted that the use of radiometry more expedient and informing at diagnostics of hyperthyrosis, than hypothyrosis.

In some cases determine absorption the red corpuscles of marked threeiodthyronine. For patients with hypothyrosis this index goes down to 8% and less.

Change of hormonal background

The most exact method of research of function of thyroidal gland is radio-immunilogical determination of thyroxin and threeiodthyronine in blood. For hypothyrosis characteristic falling of level of general thyroxin below 70 nmol/l and threeiodthyronine below 1,1 nmol/l.

For diagnostics of initial displays of hypothyrosis, conducting of differential diagnostics between the primary and second forms of hypothyrosis a necessity is determination of thyrotropine(TTH) in blood. For healthy people a concentration of him is 1,2-2,8 IU/ml. At primary hypothyrosis the level of TTH in blood is promoted to 10 times, at the second and tertiary - reduced.

By differential-diagnostic tests for differentiating of primary and second hypothyrosis tests can serve on stimulation of thyroidal gland from thyrotropine (with the use of as a marker of maintenance of thyroxin in the whey of blood, or by fascination of radio-active iodine by a thyroidal gland). Conducting of test of stimulation of hypophysis from thyroliberine (by a thyroreleasing-hormone, TRH) allows to diagnose the early forms of disease to the origin of clinical and biochemical signs, to delimit the second and tertiary forms of hypothyrosis.

Primary hypothyrosis

Initial level of T_3, T_4 is reduced, absorption of I^{131} is reduced.

Test from TTH a negative, щитоподібна gland on stimulation does not "answer" is a level of T_3, T_4 the accumulation of I^{131} remains low.

Test from TRH a negative, щитоподібна gland is irresponsive and on hypophysary, and on hypothalamic stimulation, level of T_3, T_4 the accumulation of I^{131} remains low.

Second hypophysary hypothyrosis

Initial level of T_3, T_4 is reduced, absorption of I^{131} is reduced.

A test from TTH is positive, maintenance of T_3 rises, T_4, the accumulation of I^{131} grows in a gland - a thyroid is unchanged able to answer multiplying the synthesis of hormones.

A test from TRH is negative, products of T_3, T_4 unchanging, as a hypophysis is unable to answer the increases of products of TTH on a hypothalamic stimulus.

Tertiary hypothalamic hypothyrosis

Initial level of T3, T4, TTH is reduced.

A test from TTH is positive is a level of T_3, T_4 rises, a thyroidal gland "answers" on a hypophysary stimulus.

A test from TRH is positive is a level of T_3, T_4, TTH rises, a hypophysis and thyroidal gland answer on hypophysary stimulus.

Immunological researches

For confirmation of diagnosis of primary autoimmune hypothyrosis conduct determination of antibodies to thyroglobuline. A test is considered positive at the increase of level of antibodies of more than 4 mkg/ml.

Determination of basic exchange.

A method has an auxiliary value for diagnostics of hypothyrosis and estimation of indemnification of disease on a background of substitute therapy. The indexes of basic exchange hesitate in wide enough scopes and can arrive at -60% from the proper sizes (depending on weight of disease).

Instrumental research of thyroidal gland

Computer tomography, radioisotope scanning and ultrasonic research of thyroidal gland allow visualize tissues of thyroidal gland, define reason of hypothyrosis.

The instrumental methods of research of the cardiac system serve for clarification of character of defeat of heart and haemodynamic.

Electrocardiography

The expressed of changes of EKG, as a rule, answers the degree of weight of hypothyrosis.

Typical is bradycardia. Sometimes there is deceleration of atrio-ventriculary conductivity or blockade of right foot of bunch of Gees.

Low-voltage EKG (low voltage of complexes of QRS and indents of P and T) observed in 30% patients.

Deformation of eventual part of ventriculary complex represents expressed of hypoxia of myocardium, appears two-phase indent of T, rarer by his inversion, by the decline of interval of ST below after isoline.

Echocardioscopy

Violation of phases of cardiac cycle - syndrome of hypodynamia of 1st or 2nd degree is determined. The change of central haemodynamic at hypothyrosis consists in the presence of hypokinetic circulation: the cardiac troop landings, volume of circulating blood, speed of blood stream are diminished on a background megascopic common peripheral vascular resistance.

Ultra-sound diagnostics allows to discover even the small volumes of liquid in a pericardium.

Phonocardiography

A method allows to register cardiac tones of small amplitude and low-amplitude systolic noise of muscle genesis - signs of hypothyroid heart.

Hypothyrosis primary, central, peripheral, subclinical, transitory.

1. Primary hypothyrosis - develops as a result of defeat of thyroidal gland, most frequent variant.

2. Second hypothyrosis is investigation of disfunction of hypophysis, violation of regulative and stimulant effect of hypophysary thyrotropine.

3. Tertiary hypothyrosis - observed at the diseases of hypothalamus.

4. Peripheral hypothyrosis - conditioned violation of exchange of thyroid hormones on periphery : from T_4 appears not active T_3, but that loss-active reversible RT3. The decline of

sensitiveness of receptors of tissues (resistance) is possible to the thyroid hormones. The most frequent reason is a nephrotic syndrome, cirrhosis of liver, pregnancy.

5. Subclinical hypothyrosis is a clinical syndrome, conditioned proof boundary violation of balance of thyroid hormones in an organism, at by the level of thyroidal hormonesis normal, and the level of TTH is moderately promoted. The clinical symptoms of hypothyrosis are absent.

6. Transitory hypothyrosis is observed at thyroiditis – the function of thyroid spontaneously changes from hypothyrosis to euthyrosis.

Age-old features of motion of hypothyrosis.

By the patient of sear and yellow leaf with coronal pathology and hypothyrosis, which lasted did not treat with the expressed clinical picture, a rapid medical effect is un desirable, that is why mainly treatment of thyroxine, by the combined preparations, thyroidine, but not by threeiodthyronine. Adopting thyroidal preparations strengthens the processes of metabolism in a cardiac muscle and the same can assist development or sharpening of coronal pathology, heart attack of myocardium. Development of these complications can provoke the inadequate dosage of thyroidal hormones. By such patient thyreoidine is appointed carefully, beginning from the dose of 0,01 g to the day, multiply each 5-7 days on 0,01 g under control a common feel, dynamics on EKG, level of arterial pressure. An optimum dose must, from one side, provide disappearance of most symptoms of hypothyrosis in this case, and with other - not to worsen ran across diseases of heart. Appearance or strengthening of coronal symptoms requires the immediate stopping of treatment by thyroid hormones, and after the interruption of him renew, reducing the dose of preparation.

Treatment of hypothyrosis.

Treatment at hypothyrosis begins from a valuable diet which contains the promoted amount of albumen and limited amount of carbohydrates and grew fat. In the ration of patient it must be 120-140 г squirrel. At surplus mass of body the power value of meal is limited.

At setting of course of treatment it is necessary to take into account the type of hypothyrosis (primary, secondary, tertiary), his etiology, weight of disease, eyelids of patient, presence of complications and concomitant pathology.

The basic method of treatment of all forms of hypothyrosis is the substitute therapy by preparations of thyroid hormones. The row of preparations the basic operating component of which are two hormones is used to that end – thyroxine T4 and threeiodthyronine T_3.

Threeiodthyronine - a pill contains 20 mkg or 50 mkg of T_3.

Activity of T_3 more than T_4 in 5-10 times. A biological effect is early and expressed, but the expressed and effects of sides. Threeiodthyronine has greater biological activity in 5-10 times, than thyroxine. The first signs of his action appear in 4-8 hours, maximum- on a 2-3th day, complete destroying - in 10 days. At peroral application sucked in 80-100% the accepted dose, threeiodthyronine freely penetrates through cellular membranes and operates quickly enough. Not cumulate, that is why appoint for 2-3 receptions. Speed of effect allows him to use in critical situations (hypothyroid coma). Threeiodthyronine does not use for monotherapy of hypothyrosis, as for creation of stable level him in blood frequent receptions are needed and a negative cardiotrophic effect develops quite often.

Initial dose - 2-5-10 mkg, a dose is multiplied by each 3-5 days on 2-5 mkg. A total dose can achieve 20-100 mkg.

Thyroxin- a pill contains 100 mkg of T_4.

Treatment by thyroxin more physiological, than T_3. 80% T_3 appears from T4 as a result of peripheral metabolism – monodeiodinizing of T_4. 100 mkg of T4 on a biological effect equivalently 25 mkg of T_3. Thyroxin is well sucked in gastro-intestinal system, and operating slower, deprived

many negative properties of threeiodthyronine. Preparation starts acting in 2-3 days after the beginning of reception, achieves a maximum in 2 weeks. Thyroxin is apt at cumulation- duration of action after complete abolition of preparation makes 7-10 days. As thyroxin cumulates, it is possible to accept 1 time per days.

For not young patients monotherapy of T_4 is often uneffective as a result of violation of peripheral metabolism of thyroid hormones, formation of endogenous T_3 it can be sharply reduced from exogenous T_4.

As a secretion of TTH is determined the level of T_4, thyroxin can be used for blocking of goitregenic effect of thyrostatic and for treatment of thyroiditis.

Initial dose of 10-25 mkg. A dose can be multiplied on 25 mkg each 4 weeks to achieving 100-200-400 mkg.

Thyroidine is a drop for 0,1 g and 0,05 g.

Preparation is got from the dried up fat free thyroid glands of the hammered cattle. An amount and correlation of iodine-thyronine in thyroidine considerably hesitates in the different series of preparation, orientation contains 0,1 g thyroidine, 8-10 mkg threeiodthyronine and 30-40 mkg of thyroxine. Unstable composition of preparation makes difficult his use and estimation of efficiency on the first stages of treatment, when exact minimum doses are needed. Preparation is badly sucked in from gastro-intestinal system. Cumulating, it is possible to accept 1 time per days.

Initial dose of 0,025 - 0,05 g. A dose can be multiplied each 7-10 days on 0,025-0,05 g to achieving optimum.

Thyreotom is complex preparation. Contains 10 mkg of T_3 and 40 mkg of T_4.

Thyreotom -forte - 30 mkg of T_3 and 120 mkg of T_4.

Thyreocomb is complex preparation. One pill contains 10 mkg of T_3, 70 mkg of T_4, to the 150 mg iodide of potassium.

Basic principle of treatment of hypothyrosis is based on careful and gradual, especially at the beginning of treatment, підборі of preparation and his dose. Thus determinatives is weight, duration of disease without treatment and eyelids of patient. Than heavier hypothyrosis and than longer patients (any age) were without substitute therapy, the higher their sensitiveness to the thyroid hormones, and more gradual there must be a process of adaptation. Substitute therapy of hypothyrosis is conducted during all life of patient.

Treatment of patients of young and middle ages by thyroidine without cardiovascular pathology is begun with the dose of 0,05 g on a day, gradually promoting her each 5-7 days on 0,025 g to necessary for achieving the euthyroid state.

Thyroxine begin to enter from the dose of 10-25 mkg, multiplying a dose on 25 mkg each 4 weeks (to 100-200 mkg). These preparations can be adopted 1 time per days.

Threeiodthyronine begin in the dose of 5-10 mkg on a day, a dose is promoted by each 3-5 days on 2-5 mkg, gradually taking to necessary (on the average from 20 to 100 mkg on days). Preparation is appointed for 2-3 receptions, in combination from thyroxine.

At the use of the combined preparations an initial dose is 1/4-1/8 pills, a subsequent increase is carried out slowly - once on 1-2 weeks, to achieving an optimum dose.

Thyroiditis, classification, etiology, clinical motion, diagnostics, treatment.

Thyroiditis are disease of thyroid gland, which differentiate on etiology, pathogenesis, but the obligatory component of which is inflammation.

1. Acute thyreoiditis (diffuse or local):

а) festering;

б) unfestering.

2. Subacute thyreoiditis:
а) diffuse;
б) local.
3. Chronic thyreoiditis:
а) autoimmune thyreoiditis (atrophy form);
б) fibrous invasion (of Riedel);
в) specific thyreoiditis (tubercular, syphilitic);
г) caused by physical or chemical factors;
д) caused by vermin.

Differential diagnostics of thyreoiditis is with acute clinical motion.

ACUTE FESTERING THYREOIDITIS

Acute festering thyreoiditis is festering inflammation of thyroid gland, caused a bacterial flora. Festering thyreoiditis is a rare disease.

Acute festering thyreoiditis is caused by Streptococcus pyogenes or Staphylococcus aureus, sometimes by Pneumococcus or E.coli. He arises up as complication of festering infection – acute tonsillitis, sinusitis, otitis, phlegmon, abscess, pneumonia, scarlatina, post-natal infection, more frequent without adequate antibiotic therapy.

Acute festerings regionary processes cause infecting of thyroid gland by lympho- or haematogenic way with subsequent acute inflammation of thyroid fabric. As a rule, inflammation takes an area or fate of thyroid gland fully, passes all characteristic for inflammation stages: to proliferation, exsudation, alteration.

Pyogenic changes carry more frequent local character and strike the mainly left fate (very rarely all gland). Sometimes they resolve spontaneously, abscesses are sometimes formed. Expansive destruction of thyroid gland and its capsule appears at suppuration. A process spreads for necks up to mediastinum. The function of thyroid gland usually is not violated, because the fate of gland is not staggered fully provides a necessity in thyroid hormones.

Characteristic sharp beginning: increase of temperature to 39-40 C°, tachycardia, sometimes arterial hypotension.

Pains in the area of thyroid gland are intensive, quite often have pulsating character, often there is irradiation in ears, lower or overhead jaw, back of head. Patients grumble about feeling of pressure, holding apart in the area of thyroid gland.

If for patients find out tachycardia, there is feeling of heat, it does not follow to consider them as displays of thyrotoxicose are a reaction of the vegetative nervous system on an infectiously-toxic process.

Thyroid gland at palpation sharply sickly in one of its stakes, immobile, a skin above her is red, filling out, with the local increase of temperature, very sensible. Submandibular and neck lymphatic knots are megascopic.

At spontaneous motion (especially without antibiotic therapy) in a few days after the beginning of disease festering inflammation can result in abscessing is forming of area of softening influence of thyroid gland tissue with the positive symptom of fluctuation. An abscess can spontaneously become empty on the surface of neck. The most dangerous variant of spontaneous dreange is emptying in soft tissues of neck or in mediastinum. Festering mediastinitis worsens a prognosis sharply.

Festering thyroiditis also can be complicated by the thrombosis of regional veins.

At a laboratory inspection neutrophyle leucocytosis appears with the change of leucocyte formula to the left, lymphopoenia, eosinophylopoenia.

Characteristicare dysproteinaemia, positive reaction on C-reactive protein.

At ultra-sound diagnostics in the area of defeat of thyroid gland an area appears with reduced echogenity, and on scanogramm is a ”cold knot”.

Absorption of I^{131} is not changed or reduced a thyroid gland. Scanning of gland, conducted such patient in a acute period of disease, gives a "cold" area or area with the reduced absorption of isotope.

Immunological violations at this form of thyroiditis are not observed.

For confirmation of diagnosis an aspiration biopsy is needed, the got maintenance is used for determination of sensitiveness to the antibiotics. Usually through an aspiration needle conducted instillation of antibiotics directly to gland.

Differentiating festering thyroiditis is necessary with subacute thyroiditis, by a hemorrhage in a key goitre, by sharp laryngitis, sharp unfestering thyroiditis after radial therapy.

At sub acute thyroiditis the absent is expressed a general inflammatory reaction isleucocytose, local symptomatic, multiplying of regional lymphatic knots is absent. At bleeding previous anamnesis and absence of local and general symptoms of inflammatory process is taken into account in a knot.

At thyroiditis after radial therapy pain less expressed, there are not inflammatory changes from the side of blood, in anamnesis is a course of radial therapy (usually treatment a radio-active iodine).

Laryngitis appears pain in a throat, by hoarse voice, difficulty of breathing. A diagnosis is set by laryngoscopy.

Treatment by antibiotics begins immediately, not expecting the result of microbiological research. In future penicillin can be transferable on other antibiotic, coming from the laboratory findings. If there is not information about the sensitiveness of exciter to the different types of antibiotics in the primary hearth of inflammation, it follows to appoint penicillin 500 000 IU each 4 hours during 7-10 days, in combination with sulfanylamids.

If the effect of preparations is absent or an abscess develops, surgical treatment is shown. The early section of abscess is recommended for providing of effective drenage of maintenance of abscess outside.

UNFESTERINGS ACUTE THYROIDITIS

A disease develops for as aseptic inflammation as a result of trauma and hemorrhage in a gland or after radial therapy. The common lately method of treatment the little doses of radio-active I^{131} decreased the number of radial thyroiditis. Before they met approximately in 5% cases in 2-3 weeks after adopting radio-active preparation as a result of destruction of follicle epithelium.

Patients are disturbed by pain and feeling of pressure in the area of gland, the symptoms of thyrotoxicose are moderately expressed sometimes: tachycardia, emotional weakness, ect.

Treatment is symptomatic:analgesics, β-adrenoplegics. Usually a disease passes during 3-4 weeks. It follows to pay the special attention to possibility of development of post-radiation thyroiditis at the retropectoral location of gland, as multiplying the sizes of gland as a result of inflammation can cause the compression of mediastinal organs.

SUBACUTE THYREOIDITIS

Subacute thyreiditis is an unfestering inflammatory disease of thyroid gland. Multiplying frequency of diseases appears in a fall-winter period. Women are ill in 4 times more frequent than men, age of patients can be different, however much most number of cases is on 30-40 years.

A disease is drawn a viral infection. Such facts testify about it:

- development of thyroiditis in 3-6 weeks after an acute viral respirator infection;
- a presence of prodromal phase is with a weakness, indisposition;
- an increase of morbidity of thyroiditis is during the flashes of viral infections (flu, epidemic parotitis, ect.). Penetrating inward cells, a virus causes formation of atypic albumens on which an organism reacts an inflammatory reaction. In the whey of blood of patients on subacute thyroiditis antibodies appear to the viruses of Coxaki, flu, epidemic parotitis. In reply to the damage of cells of thyroid gland an autoimmune process develops in future. The presence of immunoglobulin in tissue of thyroid gland here carries transitory character. It is marked, that subacute thyroiditis develops more frequent for the transmitters of antigen of HLA-B-35, which meets for people with a high sensitiveness to the viral diseases.

Inflammation of gland conduces to destruction of follicles and follicle epithelium, to the loss of colloid. As a result of destructive processes in blood plenty of thyroid hormones is selected which stimulates development of thyrotoxicose.

A disease begins acute from a fever, chill, general weakness, tachycardia, head pain, labouring breath, pain in the area of neck. Pain gives in ears, head, increases at the turn of head to the side. The general symptoms of inflammation prevail on occasion. At a diffuse defeat the thyroid gland is megascopic, sickly at palpation, not united with surrounding tissues, mobile, skin above herwith hyperaemia. Submandibular and neck lymphatic knots are not megascopic. Temperature of body 38-40 C°, but can be and subfebrile.

From the first days of disease growing like a weed ШОЕ appears in a clinical blood test - to 60-80 mm/h. (on occasion to 100 mm/h.) - at normal or a little promoted maintenance of leucocytes without changes in the formula of blood.

During a disease it is possible to select a few stages which different indexes of laboratory researches are during.

First (acute) stage - duration of 1-1,5 month - there is the promoted maintenance alpha-2-globulins, fibrinogen and thyroid hormones in blood at the reduced fascination of isotope of iodine by a gland. The symptoms of thyrotoxicose are clinically expressed. Such dissonance between information of scanning and clinical symptoms is explained, that the inflamed gland loses ability to fix an iodine. Maintenance of unhormonal connections of iodine, which lock fascination of iodine a thyroid gland which shows up the decline of absorption of I131, is promoted in blood. In blood act hormones are before synthesized and thyroglobuline as a result of the promoted permeability of vessels on a background inflammation.

The second stage (renewal) is duration 2,5-3 months. Violation of synthesis of hormones conduces to normalization of their level in blood, and then and to lowering of their maintenance. Болючість in a gland diminishes, there is only a sensitiveness at palpation. ШОЕ is a speed-up as well as before, the promoted maintenance is saved alpha-2-globulin and fibrinogen. Lowering of level of thyroxin and three-iod-thyronine activates the excretion of thyrotropin by hypophysis and multiplying fascination of isotope of iodine by a thyroid gland. Approximately to the end of fourth month from the moment of disease of absorption of I^{131} can be promoted at the moderately expressed clinical symptoms, dryness of skin. These phenomena pass independently, as a function of gland recommences.

The third stage (convalescence) is duration 2-4 months. The sizes of gland are normalized, pain disappears, ESR goes down, the indexes of thyroxin threeiodthyronine and thyrotropine come to the norm.

It follows to take into account that a disease is inclined to the relapse, especially under act of unfavorable factors (supercooling, overstrain, repeated viral infections).

Classification. Subacute thyroiditis is subdivided into diffuse and local. In addition, select four clinical forms of subacute thyroiditis:

- quickly making progress thyroiditis - with the acute expressed displays of inflammation;
- thyroiditis is prolonged - with slow development of symptoms;
- pseudothyrotoxic thyroiditis-with the signs of increase of function of thyroid gland;
- pseudoneoplastic thyroiditis-with the expressed compression and rapid increase of thyroid gland.

Hypothyrosis on a background of autoimmune polyendocrinopathia.

Autoimmune chronic (lymphomatose) thyroiditis is a chronic inflammatory process in a thyroid gland of autoimmune genesis. Autoimmune thyroiditis is described by Khashimoto in 1912

A disease meets for women l0-15 times more frequent, than for men. Most impressionable age-old category - 40-50 years. Autoimmune thyroiditis is most frequent from all diseases of thyroid gland.

If frequency clinically obvious autoimmune thyroiditis is 1-1,4%, the presence of antibodies to thyroperoxyde appears in 10% for practically healthy adult women and in 5% practically healthy men.

Depending on the sizes of thyroid gland and clinical picture of disease select a few clinical variants of autoimmune thyroiditis.

1. Chronic autoimmune thyroiditis. Most frequent clinical form – multiplying of thyroid gland is diffuse II or to the III degree, as a rule, without violation of function of gland, but the moderate displays of thyrotoxicose or hypothyrosis can be marked.

2. Hypertrophy form of autoimmune thyroiditis, or thyroiditis of Khashimoto. A thyroid gland is dense, diffusely megascopic. The function of gland can be not broken, but moderate violations of function appear more frequent –hypothyrosis or thyrotoxicose.

3. Atrophy form of autoimmune thyroiditis. In the moment of inspection the volume of thyroid gland is not megascopic, in anamnesis there is not a goitre or the moderate increase of thyroid gland was before marked. Functionally – hypothyrosis.

Some authors consider hypertrophy and atrophy forms by the successive phases of the same process.

Grounding of diagnosis of autoimmune thyroiditis.Clinic

Thyroiditis Khashimoto develops gradually. During the first years of disease of complaint and symptoms, as a rule, absent.

1. Squeezing symptoms.

At the hypertrophy form of autoimmune thyroiditis a thyroid gland is megascopic, at an atrophy form sizes of her in a norm or even diminished.

At the hypertrophy form of autoimmune thyroiditis basic complaints are related to multiplying a thyroid gland: feeling of discomfort at swallowing, laboured breathing, hoarse of voice, feeling of foreign body in a throat, quite often insignificant pain or feeling of discomfort, pressure in the area of front surface of neck.

A gland is megascopic symmetric, dense consistency. At palpation “inequality”, “knotted” of surface, its different closeness and elasticity, is determined. Appearance of expressive knotted of gland is possible in future.

Characteristic «symptom of waggle» - by palpation of one part of gland the other waggles the other part.

At the atrophy form of autoimmune thyroiditis gland is diminished, fibrosis develops in her. Thyroid gland or it is impossible to find, or its sizes within the limits of I degree of increase.

A focal form is characterized the defeat of one part of thyroid gland. This part is little, dense. In course of time dense knots can appear in thyroid gland (key form of autoimmune thyroiditis).

2. Change of the functional state of thyroid gland.

For patients on autoimmune thyroiditis is possible different variants of the functional state of thyroid gland. Hypothyrosis is clinically expressed observed in 36% inspected patients; subclinical hypothyrosis, diagnosed laboratory – in 40%; hyperfunction of thyroid gland – in 4%, in 19% patients – euthyroid state.

The hypertrophy form of thyroiditis in the first years of disease can run across with the phenomena of thyrotoxicose, which is, as a rule, easy or middle weight. Thyroiditis with the clinical picture of thyrotoxicose, as a rule, meets in a few first years of development of disease and conditioned the presence of thyroid-stimulating antibodies at the enough body of tissue of thyroid gland which saved normal structures and able to answer increase functional activity on the indicated antibodies. The subjective displays of disease are effaced: general weakness, fatigue, palpitation, crabbiness.

The level of endogenous thyroid hormones is sometimes promoted provided their passive exit from staggered the destructive autoimmune process of follicles of thyroid gland. In any event thyrotoxicose carries temporal character. Thyrotoxicose at autoimmune thyroiditis ("Khashitoxicose") appears by palpitation, feeling of heat, crabbiness, appearance of ophthalmopathia is possible.

The decline of function of thyroid gland is possible at both forms of autoimmune thyroiditis. Multiplying mass of body, sensitiveness to the cold, dryness of skin and fall of hairs, decline of memory, constipations, bradycardia, violation of sexual functions appears thus.

Very rarely there is a «turn» of function of щитоподібної gland, when present during a few years hypothyrosis as a result of autoimmune thyroiditis changes on the hyperthyroid state. Such dynamics of the functional state of gland is the result of change of orientation of autoimmune process – change of titles of thyroid-stimulating antibodies and antibodies which lock binding of TTH to his receptor. The obligatory condition of development of hyperthyrosis is a presence of enough body of follicle tissue of thyroid gland which is able to synthesize the promoted amount of thyroid hormones.

Additional researches

Multiplying ESR is determined in a clinical blood test, at development of hypothyrosis is anaemia.

In biochemical tests is multiplying maintenance of sial acids, gamma-globulins, fibrin; at development of hypothyrosis –hypercholesterinaemia, multiplying maintenance of β-lipoproteins.

Immunologically multiplying maintenance appears at blood of T-helpers, immunoglobulins, appearance of circulating immune complexes, decline of number of T-supressors.

Typical is presence in the whey of blood of antibodies to the different components of thyroid gland -thyroglobulin, peroxydasa, thyroid-stimulating antibodies, thyroid-depressing antibodies, antibodies to the thyroid hormones and other. Frequency of exposure of auto-antibodies depends on the sensitiveness of the used methods of determination.

The title of antibodies in the whey of blood correlates autoimmune answers with activity.

Antibodies to thyroglobulin and thyroperoxydasa by the radioimmunological method appear in 100% cases at a hypertrophy and in 94%-100% at atrophy forms of autoimmune thyroiditis.

If title of antibodies to thyroglobulin in the whey of blood 1:100 and higher, and title of antibodies to thyroiperoxydase higher 1:32, it is no doubt possible to say, that in patient patient on autoimmune thyroiditis. In this case there is not a necessity in the biopsy of thyroid gland. For finding out of diagnosis at the doubtful titles of antibodies to thyroid gland is shown in blood.

At autoimmune thyroiditis appear also thyroid-stimulating antibodies, and in 2-5% patients he is combined with a diffuse toxic goitre (Khashitoxicose). At scanning in such cases next to low absorption of iodine the thyroid gland are areas with his high accumulation.

At "Khashitoxicose" maintenance of thyroxin and threeiodthyronine rises in blood, at hypothyrosis their maintenance goes down. Highly sensitive methods of determination of level of thyroid hormones, and also conducting of test from thyroliberine allow to find out violation of function of thyroid gland on the most early stages.

An increase of level of TTH in the whey of blood is the most early diagnostic sign of hypothyrosis, when his clinical symptoms are yet absent. The concentration of TTH in the whey of blood of over 5 mkIU/ml (norm – 0,4-4,5 mkIU/ml) at normal maintenance of free T_4 testifies to subclinical hypothyrosis. Presence of clinical signs at the promoted level of TTH and during the reduced concentration of free T_4 is a sign of manifest or clinical hypothyrosis.

Table of contents of protein-connected iodine in the whey of blood of patients on autoimmune thyroiditis can be promoted at the normal level of T_4. It is related to that at this disease the amount of NCI (nono-calorigenic iodineproteids) is multiplied and the synthesis of thyroxin goes down. At autoimmune thyroiditis grows amount other iodine-contents albumens. The defect of organification of iodine is confirmed a test with perchlorate of potassium. The level of iodine-thyrosin rises in blood; sometimes it unites with multiplying maintenance of T_3 in the whey of blood at euthyroid or even hypothyroid clinical state of patients.

Tests with oppressing the synthesis of T_3 and tests with stimulation by thyroliberine can be negative. It goes to show that activity of thyroid gland is under control not TTH, but thyroid-stimulating immunoglobulins. Absence in such cases of thyrotoxicose is explained by destruction of more greater part of functioning tissue of gland.

Absorption and accumulation of iodine^{131} in most cases meets standard or it is diminished.

In the first months of disease on occasion the increase of absorption of iodine (in the case of previous sharp decline of supplies of iodine in a gland) and high level of protein-connected iodine appears in blood. On scanogramm at a hypertrophy form a thyroid gland is megascopic, the contours of her are unclear, a form is changed (not form of "butterfly", but asymmetric), the accumulation of radiocontrastpreparation is uneven, the mosaic of picture is determined, there are areas of the reduced accumulation, in a center there is not intensive fascination which can give the picture of "multiknot goitre", by palpation knots are not determined although.

On the measure of progress of fibrin processes and development of hypothyrosis absorption of radio-iodine and maintenance of protein-connected iodine goes down. At an atrophy form the sizes of gland are diminished, at a knot form "cold" knots are determined. Variants are however possible with a normal or even promoted accumulation (due to multiplying mass of gland) on a background the clinical symptoms of hypothyrosis.

Sonography or ultrasound research of thyroid gland allows to define an increase or diminishing of its sizes (to the volume of gland in ml). For autoimmune thyroiditis characteristic diffuse decline her echogenity, the uneven structure of thyroid gland is determined with the presence of hypoechogenic areas or knots. The similar picture takes place and at a diffuse toxic goitre, that is why from data of ultrasound diagnostics it is impossible to put a final diagnosis.

Stages of autoimmune thyroiditis from information of sonography:

1) multiplying all sizes of thyroid gland is with the proper increase of volume of gland;

2) uneven decline of echogenity of parenchyma, which can be more or less even or (more frequent) has "pointed" character.

3) a compression of capsule of gland is as a hyperechogenic contour of both stakes;

4) appearance of shallow dense linear structures, located chaotically (areas of fibrosis).

Sonographic sign of considerable duration of disease:

1) the compression of capsule of gland is more expressed;

2) multiplying an amount and sizes of dense linear structures which gain character of layers - prolonged the dense linear including form of "pseudopartial" echostucture of gland.

Diffuse changes of parenchyma of thyroid gland at autoimmune thyroiditis it is impossible to name strictly specific.

The decline of echogenity of parenchyma can be observed at diffuse toxic and euthyroid goitre, shallow hypo- and anechogenic including – at subacute thyroiditis, linear dense structures as displays of fibrous changes – in general at any variants of diffuse pathology of thyroid gland. For differential diagnostics in every case it is necessary to take into account the aggregate of these signs and degree of expressed each of them separately.

Autoimmune thyroiditis however formed a diffuse autoimmune disease of thyroid gland.

"Pseudoknot" echographic variant of autoimmune thyroiditis: focal lymph-plasmacytary infiltration, degenerative changes of follicle epithelium with expansion of follicles, hypertrophy of separate particles with the fibrose changes of stroma can create the picture of “pseudoknots” on a background the typical displays of autoimmune thyroiditis.

«Pseudoknot» appear on a background the expressed heterogeneity of echostructure of parenchyma- rounded, wrong form hypoechogenic elements with clear contours, which imitate knot structures. On the apparate of middle class it is possible to find out focal elements with size about 2-3 mm.

Term a "knot" foresees the presence of the encapsulated education which expressly differs for echostructure from surrounding fabric. Morphological substrate of «knot» can be a colloid proliferating goitre, knot or tumor.

At autoimmune thyroiditis morphological substrate of “pseudoknots” are the extended follicles, hearths of lymphoid infiltration by a size about 2-3 mm and anymore. Cytological verification of such elements on technical reasons is laboured. Combination of knot goitre from autoimmune thyroiditis is fully possible, and in these cases better not to unite two on a creature different disease in one diagnosis, but conduct, it is possible, clinical, pathomorphological and echographic differentiation of present changes. This is important for the adequate choice of subsequent tactic of conservative treatment with periodic control of function of gland or operative treatment.

It is considered that malignant new formations (lymphoma) on a background autoimmune thyroiditis very rare.

Long time chronic autoimmune thyroiditis on sonogram appears the acute diminishing of all sizes and volumes of thyroid gland, strengthening of heterogenety of echostructure due to the presence of the numerous expressed linear dense structures which occupy practically all parenchyma of gland.

If there is clinical information about possibility of malignant regeneration of thyroid gland (development of knot elements), the thin-needle biopsy of suspicious area is needed, without regard to the presence of high title of antibodies. A presence at the patient of the phenomena of thyrotoxicose does not eliminate possibility of malignant regeneration of thyroid gland also.

Forms of goitre. Differential diagnosis of goiter.

It follows to differentiate an endemic and sporadic goiter, chronic autoimmune thyroiditis, fibroplastic goiter of Riedel, cysts of neck, tumor and cyst of mediastinum, malignant new formations of thyroid gland and metastases of malignant tumors in lymphoid knots of neck.

About the presence of goiter Khashimoto testifies the high titre of auto-antibodies to tissue of thyroid gland, mosaic structure of thyroid gland at scanning, diminishing of sizes of goiter at a prednizolon test.

A goiter of Riedel is dense uneven element, connected with inferior tissues, but it is not fixed to the skin.

The cysts of neck, unlike goiters, are not displaced at swallowing motions, does not accumulate a radio-active iodine.

Intrapectoral goiters, unlike tumors, are displaced at swallowing and cough, accumulate a radio-active iodine, have clear contours on sciagrams.

At malignant new formations of thyroid gland a radio-active iodine accumulates very poorly or does not accumulate quite, on 2nd the stage of disease metastases appear in regional lymphoid knots, on 3rd the stage - metastases are remote.

Complication at an endemic and sporadic goiter:

- hemorrhages in a goiter:
- calcification of goiter:
- inflammation;
- malignant regeneration.

Differential diagnostics of autoimmune thyroiditis is conducted with a knot and mixed euthyroid goiter:, cancer of thyroid gland, diffuse toxic goiter, fibrose thyroiditis of Riedel.

There are not signs of hypothyrosis at a knot or mixed euthyroid goiter:, characteristic for thyroiditis of Khashimoto. On scanogramm and thyrolymphogramm the defects of accumulation of isotope are determined, the low title of auto-antibodies appears and the morphological picture of thyroiditis is absent in bioptate.

The stony closeness of gland, absence of mobility, multiplying regional lymphatic knots, asymmetricness of scanographic silhouettes, presence of "cold" areas, unequal contours of gland, low titre of auto-antibodies, testifies about the cancer of thyroid gland. Under act of threoidine and prednizolon patients on thyroiditis have diminishing of sizes and closeness of goiter.

Undulating motion of disease and other expressed symptoms of diffuse toxic goiter, results of the special methods of inspection, allow to diagnose autoimmune thyroiditis. At a diffuse toxic goitre after stimulation by thyroliberine initial the normal level of thyreotropine does not give an increase.

To differentiate fibrose and autoimmune thyroiditis help characteristic for autoimmune thyroiditis sign ofhypothyrosis, moderate closeness of goiter, high titre of auto-antibodies, histological picture.

A disease has a tendency to slow progress. On occasion a satisfactory feel and capacity of patients is saved during 15-18, without regard to the brief sharpening. In the period of sharpening of thyroiditis there can be the phenomena of insignificant thyrotoxicose or hypothyrosis; the last more frequent meets after births.

As a rule, autoimmune thyroiditis does not give a malignant regeneration. But combination of autoimmune thyroiditis with the cancer of thyroid gland meets in 1-15% patients. Exceptionally rarely there is lymphoma of thyroid gland.

Cases are described, when by patients on autoimmune thyroiditis from hypothyrosis spontaneous the remission develops in the period of pregnancy. However sometimes for patients on euthyroid autoimmune thyroiditis hypothyrosis developed after births.

Control of initial level of knowledges

Task 1

What from Iodine-content hormones is of thyroid hormonal non-active?

a) general T_4;
b) free T_4;
c0 general T_3;
d) free T_3;
e) **rreversible T_3**

Task 2
What dose of thyreoidine does it follow to begin treatment of hypothyrosis for patients on IHT?
a) 0.025g time per days);
b) 0.05g 1 time per days,
c) 1g 1 time per days,
d) 0.1g 2 times per days
Task 3
What disease or state is deceleration of time of Achyl reflex most expressed at?

a) saccharine diabetes;
b) atherosclerosis;
c) Hypopotassiumaemia;
d) 100-primary hypothyrosis

Task 4
What from preparations more rationally to appoint for a patient with autoimmune thyreoiditis at the acute increase of thyroid and increase of title of antithyreoid antibodies?

a) thyroxin;
b) prednizolon;
c) suprastinum;
d) verapamilum

Task 5
By which from tests is differential diagnostics conducted between primary and secondhypothyrosis?
a)determine the level of T_3 and T_4 of blood;
b) determine the level of TTH in blood;
c) test with thyroliberine

Task 6
What research most informative during conducting of differential diagnostics between secondary and tertiary hypothyrosis?
a) detyermine the level of TTH in blood;
b) test with thyroliberine

Task 7
A sensitiveness to the thyroidal hormones is most promoted:
a) at child's age;
b) during pregnancy;
c) at declining years;
d) by decompensations of cardiac activity

Task 8
Overdosing of thyroid hormones is accompanied by:

a) tremor;
b) promoted excitability, violation of sleep;
c) nightly sweat; 80-decrease the masses of body;
d) all of the higher resulted signs

Task 9
Most reliable complication at births for pregnant with hypothyrosis:

a) weakness of childbirth;
b) swift births;

c) haemorrhagia at births;
d) cardiac insufficiency

Task 10
The consequences of influencing of isotopes of radio-active iodine are remote on a thyroid as a result of failure of step of Chernobyl nuclear power-station:
a) thyroiditis;
b) hypothyrosis;
c) cancer;
d) all is higher marked

Task 11
How does pregnancy influence on motion of grade of thyreotoxicosis?
a) negative;
b) positive;
c) without influences

Task 12
What changes of general analysis of blood can appear at hypothyrosis?
a) anaemia;
b) lymphocytosis;
c) eosinophylia;
d) all is listed above

1	2	3	4	5	6	7	8	9	10	11	12
E	A	D	B	B	B	D	D	A	D	B	D

Control of eventual level of knowledges

Task 1
The syndrome of Schmidt includes:
a) hypocorticism+hypothyrosis+hypogonadism;
b) hypothyrosis+hypogonadism;
c) hypocorticism + hypogonadism;
d) galactorrhea + hypothyrosis

Task 2
Characteristic for hypothyrosis is the promoted level of TTH in blood after birth of child during:
a) 12 hours;
b) 24 hours;
c) 48 hours;
d) more than 48 hours;

Task 3
By main reason of galactorrhea at hypothyrosis it is:
a) deficiency of thyroid hormones;

b) remain ofthyrotropine;
c) hypersecretion of thyroliberine;
d) all is marked

Task 4
The level of TTH is promoted in the whey of blood does not testify in behalf of hypothyrosis in cases:

a) surplus secretion of TTH by a tumor of the hypophysis;
b) ectopic secretion of TTH;
c) new-born to 48 hours after birth;
d) all above is marked

Task 5
What from criteria most adequate in the correction of dose of thyroidal hormones at treatment of Khashimoto disease?

a) normalisation of maintenance of T3;
b) normalisation of maintenance of T4;
c) normalisation of TTH;
d) decrease of sizes and closeness of thyroid;
e) decrease of title of antibodies to thyroglobulin.

Task 6
Major feature of structure of ultrasonic image of thyroid at autoimmune thyroiditis:

a) echogenic closeness is promoted;
b) echogenic closeness is reduced;
c) echogenic closeness is not changed;
d) echogenic closeness is unhomogeneous;
e) echogenic closeness is homogeneous

Task 7
What type of immunity can be violated at autoimmune thyroiditis?

a) humoral;
b) tissue;
c) both of them

Task 8
A basic testimony is for setting of riphatyroine:
a) primary hypothyrosis;
b) secondary hypothyrosis;
c**)** tertiary hypothyrosis

Task 9
What is the maximal duration of action of thyroxine after his abolition?

a) one day;
b) two days;
c) 4-6 days;
d) 2-3 weeks;
e) 1 month

Task 10
By the most critical period for normal development of cerebrum of child, when the sufficient level of thyroidal hormones is needed:

a) first months after birth;
b) first year;
c) a period from 3 to 5 years old;
d) a period from 5 to 10 years old

Task 11
To the patients which have a hypothyroid form are contra-indicated:

a) thyroxine;
b) corticosteroids;
c) threeiodthyronine;
d) artificial ventilation of lights;
e) active warming

Task 12
Hypothyrosis is characteristic by presence of symptoms:
a)bradycardia;
b) hypotonia;
c) amenorrhea;
d) hypomymia;
e) all is higher marked

1	2	3	4	5	6	7	8	9	10	11	12
B	C	A	D	E	C	C	C	D	C	E	E

Test tasks

Task 1
What is characterized the syndrome of Schmidt?

a) hypothyrosis and hypocorticism;
b) hypothyrosis, hypocorticism and saccharine diabetes;
c) hypothyrosis and hypercorticism

Task 2
What does characterize the syndrome of Van-Vick-Ross-Genss?

a) hypothyrosis and saccharine diabetes;
b) hypothyrosis and lactorrhea–amenorrhea;
c) hypothyrosis and hypocorticism

Task 3
Name a major testimony for setting of glucocorticoids at treatment of autoimmune thyroiditis:

a) largenesses of goitre;
b) large closeness of goitre;
c) signs of inflammation;

d) knots.

Task 4.
All below transferred illnesses have an autoimmune origin, except for:
a) thyroiditis of Hashimoto;
b) Adison`s disease;
c) syndrome of Nelson

Task 5
What type of therapy does advantage give oneself up in treatment of Ridel`s goitre?
a) radiation;
b) surgical;
c) ubstitute therapy by thyroid hormones;

Task 6
In basis of pathogeny of hypothyroid coma lies:
a) supression of respiratory center and hypoxia of brain;
b) making progress decline of the cardiac troop landings;
c) hypothermy and growing hypocorticism;
d) all is higher marked

Task 7
Early post-operation hypothyrosis is conditioned:
a) decrease feed cult of thyroid;
b) surplus delete of thyroid;
c) autoimmune defeat of tissue cult

Task 8
Early post-operation hylothyrosis develops:
a) on draught of the first 6 months after operation;
b) 9 months;
c) 12 months

Task 9
Early post-radio-iodine hypothyrosis develops not later:
a)-9 months;
b) 1 year;
c) 2 years

Task 10
The early symptom of hypothyrosis is:
a)noticeable increase of mass of body;
b**) deafness;** c) bradycardia;
d) shelling of skin

Task 11
What of thyroid preparations does it follow to give advantage to at treatment of hypothyroid coma?
a) thyroxin;
b) threeiodthyronin;

c) thyronin

Task 12
What from criteria most informing for the estimation of efficiency of substitute therapy of primary hypothyrosis?
a) concentration of cholesterol of blood;
b) Achyll reflex; 70- concentration of T_3 and T_4 in blood;
c) concentration of TTH in blood

1	2	3	4	5	6	7	8	9	10	11	12
B	B	C	C	B	C	A	A	A	B	B	C

Questions for control to the theme

1. Determination, etiology, clinical manifistations, diagnosis and treatment of iodine deficiency.
2. Determination of concept of hypothyrosis(HT).
3. Etiology of hypothyrosis.
4. Metabolic violations at hypothyrosis.
5. Clinical displays of HT.
6. The state of thyroid at HT.
7. Change of the state of skin and hypodermic cellulose.
8. Symptoms of Baer and Hertog.
9. Hypothyroid myopathy.
10. Myasthenic and myotonic syndromes at HT.
11. Syndromes of Hoffmann at HT.
12. A violation of the peripheral nervous system at HT–polyneuropathy.
13. Psycho-emotional violations are at HT.
14. Changes of ear and articulations at HT.
15. A violation of the cardiac system at HT.
16. Signs of «hypothyroid» heart.
17. Changes of pulse and arterial pressure at HT.
18. Displays and reasons of hydropericardium, polyserositis at HT.
19. A violation of bones and joints at HT.
20. A violation of HT.
21. A defeat of kidneys at HT.
22. Disfunction of breathing organs at HT.
23. Thyrogenic anaemia at HT.
24. Endocrine violations at HT.
25. Changes of haemogramm at HT.
26. Biochemical violations at HT.
27. Violation of iodine balance at HT.
28. Radiometry of thyroid gland and radioisotope scanning of thyroid gland.
29. A change of hormonal background and tests of TTH and TRH at primary HT.
30. A change of hormonal background and tests of TTH and TRH at secondary HT.
31. A change of hormonal background and tests of TTH and TRH at tertiary HT.
32. Determination of peripheral HT.

33. Determination of subclinical HT.
34. Age-old features of motion of HT.
35. Treatment of hypothyrosis.
36. Description of preparations which contain typhoid hormones.
37. Determination of thyroidites(T).
38. Classification of T.
39. Thyroiditis with acute clinical motion.
40. Thyroiditis on a background of autoimmune polyendocrinopathia.
41. Determination of chronic autoimmune thyroiditis (ChAT).
42. Clinical variants of ChAT (hypertrophy, atrophy, ect.).
43. Squeezing symptoms at ChAT.
44. Change of the functional state of thyroidal gland.
45. Informing of laboratory and instrumental researches at ChAT.
46. Immunological research in diagnostics of ChAT.
47. Indexes of iodine balance for patients on ChAT.
48. Change of radiometry and radioisotope scanning at ChAT.
49. Sonography of sign of ChAT.
50. Key forms of goitre, determination.
51. Differential diagnostics of key forms of goitre.

Practical tasks to topic.

1. To define character of changes of thyroid with the help of palpation.
2. To define the factors of risk of HT, the etiologic factors of disease are possible; retrospectively to set the initial signs of disease, estimate adequacy of applied earlier diagnostic measures; to set pharmacological anamnesis; to define the basic stages of motion of disease.
3. To ground the diagnosis of HT.
4. To set character of complications of HT.
5. To estimate the results of clinic-laboratory and instrumental researches.
6. To define the degree of weight of HT.
7. To conduct the differential diagnosis of thyroid goitre.

Protocol of the clinical examination of the patient

Name, surname of the patient_____
Age_____ **Profession**_____

Complaints of the patient_____
_____
_____
_____
_____
_____

Anamnesis morbi
_____
_____

Last exacerbation_____

Anamnesis morbi

Results of the physical examination:

Preliminary diagnosis:

Plan of investigation:

Results of the additional methods of investigations:

Rationale of the clinical diagnosis:

_____
_____
_____
_____
_____

Clinical diagnosis:
Main disease

_____
_____
_____

Accompanying disease

_____
_____
_____

Complications_____
_____
_____
___

Treatment:
1. Regime_____
2. Diet_____
3._____
4._____
5…….

literature :

1. Dwarfism: Medical and Psychosocial Aspects of Profound Short Stature. Betti M, Aelson. 2005.-368 p.
2. Endocrinology and metabolism /Ed. by Pinchera. - London: McGraw Hill Int., 2001. -811p.
3. Handbook of Physiology. Section 7: Endocrine system. Volume III: Endocrine regulation of Water and electrolyte balance. / Ed. by J.C. S. Fray. - Oxford University press, 2000.-750 p.
4. Textbook of endocrine physiology / Ed. by J.E. Griffin, S.R. Ojeda. - 4-th ed. -Oxford University press, 2000. - 490 p.
5. Thyroid Disorders. Mario Skodur, Jesse B. Wilder. - Cleveland Clinic Press, 2006. - 224p.

Інформаційні ресурси

сайт кафедри внутрішньої медицини № 3 ХНМУ http://www. vnmed3.kharkiv.ua/, встановлене інформаційно-освітнє середовище Moodle на піддомен сайта http://distance-training. vnmed3.kharkiv.ua

Методична вказівка складена:

Методична вказівка переглянута і затверджена на засіданні кафедри: _____

З доповненнями (змінами) _____

Завідувач кафедри Л.В. Журавльова