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Diagnostic Aspects and Comparative Diagnostics of Thyroid Disease

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Abstract: The problem of timely detection of endocrine pathology remains relevant. When analyzing the incidence and prevalence of diseases of the endocrine system, nutritional disorders and metabolic disorders, it was revealed that the prevalence of these diseases in terms of attendance is 32.8%, according to dispensary records (form 30) - 18.8%, according to the results of in-depth examinations - 127, 4‰ [1]. In different regions up to 40-55% of all endocrine pathology is due to diseases of the thyroid gland. Therefore, every doctor faces the problem of early diagnosis and differential diagnosis (DD) of thyroid diseases.

Key points: Diffuse toxic goiter, thyroid cancer, thyroiditis, metastases.

Introduction. To assess the condition of the thyroid gland, it is important to determine its size, position, consistency, structure, mobility, and function. The thyroid gland is mobile when swallowing, but the same is observed with palpable adenomas of the parathyroid glands (pseudostrum). Dermoids, cysts, lymphomas do not move during swallowing movements. The morphological characteristics of the thyroid gland, along with palpation, are revealed by scinti- and echography. Radionuclide techniques detect areas of increased (“hot” nodes) or decreased (“cold” nodes) assimilation of the technetium-99 isotope. In this case, ectopic nodes, retrosternal position of the gland, and additional lobules are also identified [2]. Echographically reliably assess the size of the organ, its organotopy, compaction of the parenchyma (in chronic thyroiditis) or hypoechogenicity (acute, immune thyroiditis, chronic lymphocytic), the presence of nodes and their characteristics. Hypoechoic nodes are found in gland cancer, anechoic with the phenomenon of sound amplification typical for fluid cysts (fluid node). Echo-positive nodes are observed with cell proliferation (adenomas). High-density formations with the phenomenon of acoustic shadowing are pathognomonic for calcification. Thyroid hormones increase protein synthesis in all tissues of the body. T3 increases oxygen utilization by activating Na+, K+-ATPase (Na-pump), primarily in the heart, liver, kidneys and skeletal muscles. When determining thyroxine (T4) and triiodothyronine (T3), it should be remembered that they reflect an inactive pool of hormones, 99.95% bound to protein (thyroid binding globulin = TSH). Therefore, to exclude the influence of protein content on hormone levels, it is necessary to determine free thyroxine (the norm is 10-26 pmol/l). Hyperthyroidism with an isolated increase in T3 is possible; the concentration of its free fraction in the serum is normally 1.2-3.1 nmol/l). DD of primary and secondary hypothyroidism is based on determining the level of thyroid-stimulating hormone (TSH). The most important is a test using a thyroid-stimulating releasing factor to determine the TSH level before and after prescribing the releasing factor. The difference must be at least 25 mU/l. The test serves for early diagnosis of

disrupted pituitary-gland connections; it is applicable for Graves' disease, autonomy of the thyroid gland and early differential diagnosis (DD) of primary and secondary hypothyroidism.

Thyroid tumors

Most thyroid nodules are benign. Thyroid cancer is often relatively differentiated, not characterized by high malignancy, but in young people it is prone to unfavorable flow. Usually the first sign noted by a doctor or the patient has a nodule in the thyroid gland, less often - a small tumor of the thyroid gland debuts with metastasis in the lymph nodes, lungs or bones.

Suspicion of thyroid cancer increases with the following factors:

1. Age. Cancer is more likely in young people.
2. Gender - male. In women, cancer occurs 2 times more often than in men, but thyroid diseases are also more common in women (8:1).
3. Single knot. Multiple nodes are usually observed in benign forms.
4. “Cold node” during isotope scanning. A “hot node” is a sign of a benign formation.
5. Anamnestic information about irradiation of the neck, occipital region, thymus gland, especially in childhood and adolescence.
6. Ultrasound or X-ray examination reveals delicate “lace” calcification (papillary carcinoma) or dense homogeneous calcification (medullary carcinoma).
7. Rapid increase in education.
8. A node of stony density, its adhesion to the skin, decreased mobility of the gland.

Clinically, Horner's syndrome quickly develops with pain radiating to the ear and back of the head, roughness, hoarseness or hoarseness of the voice due to paresis of the recurrent nerve, later a painless increase supraclavicular and cervical lymph nodes. Among carcinomas, relatively differentiated iodine-accumulating forms predominate (65%). 33% account for prognostically unfavorable undifferentiatedforms. Rarely (1%) medullary carcinoma (C-cell) is observed, occurring with an increase in calcitonin levels, especially after the pentagastrin injection test. In the clinic, persistent diarrhea predominates. C-cell adenocarcinoma is combined with neurocutaneous syndrome (or Cohen's syndrome), is characterized by a clear familial concentration, and is combined with other adenomas, neuromas and pheochromocytoma. The remaining 1% accounts for carcinomas arising from squamous epithelium, hemangioendothelioma, teratoma and lymphoma, undifferentiated tumors. Distant metastases are found primarily in the lungs and bones. DD of thyroid tumors is carried out with metastases of extraglandular neoplasia in its parenchyma, with nodular forms of thyroiditis. After Scintigraphy, which reveals the degree of accumulation of the isotope in the node (cold or hot node), and echography, which determines its density and structure, shows a puncture biopsy (diagnostic value - 92%), which decisively classifies the tumor into a classification category, determining therapeutic tactics and prognosis. If, with negative results, clinical suspicion of a tumor remains, then one has to undergo puncture of various areas of the suspicious node or open surgery. During dynamic monitoring of irradiated patients or those previously operated on for gland tumors, in the presence of non-tumor nodes, determination of the level of thyroglobulin and calcitonin (tumor markers) is indicated. The determination of calcitonin, as opposed to thyroglobulin, is also applicable for the primary diagnosis of thyrocarcinomas.

Thyroiditis is divided into acute, subacute and chronic. Acute thyroiditis with neutrophilic infiltration (purulent) is caused by strepto-, staphyloid and pneumococci. The primary focus is most often in the oral cavity. In the clinical picture, local signs of inflammation come to the fore: swelling, swelling of the gland, redness of the skin over it, its pain on palpation, and sometimes fluctuation. Common reactions include increased temperature, malaise, accelerated ESR, neutrophilic leukocytosis with a shift to the left. High-dose radiotherapy for non-thyroid malignancies leads to very painful thyroiditis. The likelihood of hypothyroidism after this is very high and thyroid function should be assessed every 6-12 months. Thyroiditis can be caused by

amiodarone and α-interferon. In non-purulent acute thyroiditis, the clinical picture is much milder, but reliable DD is clinically impossible; a puncture biopsy is required. With the described variants of thyroiditis, the function of the gland does not change. Subacute (giant cell) thyroiditis is known as Querfein's thyroiditis. Histologically, a moderate lymphocytic infiltration is determined, reminiscent of Hashimoto's thyroiditis and "silent lymphocytic thyroiditis", but its typical combination is with giant cells, polymorphonuclear leukocytes and rupture of follicles (the latter leads to the release of hormones and the development of thyrotoxicosis relatively early from the onset of the disease). Gynecotropic (5:1). They are usually associated with overt or asymptomatic viral infection. General symptoms predominate in the form of low-grade fever, fatigue, pain radiating to the lower jaw, chin, chest, back of the head, ear, and a feeling of dry throat. The transition of pain from one side to the other and back, its intensification when tilting or turning the head, is very typical. The gland is tense. Signs of moderate thyrotoxicosis are determined: weight loss, nervousness, heat intolerance. ESR is accelerated, leukocytosis is moderate, alpha-2-globulinemia is detected. The maximum brightness of symptoms lasts 1-3 months, relapses are possible, in between which moderate hypothyroidism is determined. I131 accumulation is reduced,antibodies to the thyroid gland are not detected or their titer is low. After recovery, the function of the gland is usually completely restored, but there may be outcomes in hypothyroidism (10%). The nature of pain in Querfein's thyroiditis sometimes serves as a reason for the erroneous diagnosis of pharyngitis, otitis, and dental pain. Chronic thyroiditis is divided into lymphocytic (autoimmune = Hashimoto's), fibrous (Riedel's struma) and specific (syphilitic, tuberculous). Hashimoto's lymphocytic thyroiditis is an autoimmune thyroiditis with a high concentration of circulating antibodies to thyroglobulin, thyroid microsomes and colloid. In isolated cases, familial variants and autosomal dominant (AD) transmission have been described. The disease is very common, observed mainly in women (8:1). It often accompanies other autoimmune diseases of the endocrine glands (Addison's disease, etc.) and chromosomal pathology (Down's disease, Kleinfelter's, Turner's syndromes). The disease begins gradually. The gland is dense, painful, the pain radiates to the ears. The size gradually increases, difficulty swallowing appears. The temperature is not changed, the ESR is increased. The metabolism is initially hyperthyroid, then euthyroid, and can eventually become hypothyroid. The titers of antimicrosomal antibodies are significantly increased. Decisive for diagnosis is a puncture biopsy (lymphocytic infiltration of the gland parenchyma). The clinical picture is very poor, thyroiditis often occurs subclinically, so Hashimoto's thyroiditis is often diagnosed retrospectively by the development of hypothyroidism or struma. Increased likelihood of papillary carcinoma and thyroid lymphoma. It is possible that one of the variants of Hashimoto’s disease is the so-called. painless silent thyroiditis (silentthen - euthyroid and may eventually become hypothyroid. The titers of antimicrosomal antibodies are significantly increased. Decisive for diagnosis is a puncture biopsy (lymphocytic infiltration of the gland parenchyma). The clinical picture is very poor, thyroiditis often occurs subclinically, so Hashimoto's thyroiditis is often diagnosed retrospectively by the development of hypothyroidism or struma. Increased likelihood of papillary carcinoma and thyroid lymphoma. 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After a new pregnancy, the condition recurs. Antibodies to thyroglobulin are detected in the blood Chronic fibrous thyroiditis of Riedel occurs with a sharp thickening of the gland and is very rare. The fibrotic process can lead to narrowing of the trachea with the development of stridor. Riedel's thyroiditis may be one of the manifestations of retroperitoneal fibrosis (Ormond's disease). DD with malignomas is carried out

based on the results of a puncture biopsy. Euthyroid struma is the most common variant of all struma. If it occurs in a population with a frequency of 10%, then they speak of endemic struma; with a lower prevalence, struma is considered sporadic. Euthyroid struma is a non-inflammatory non-tumor diffuse or nodular enlargement of the thyroid glandwithout signs of hyper- or hypothyroidism. The reason is iodine deficiency, the need for which, according to WHO, is 50-100 mcg/day for children 1 year old, 100 mcg/day for children 2-10 years old, and 150-200 mcg/day for children over 10 years old. Sporadic struma develops with hereditary defect in iodine absorption, administration of lithium, iodine, thyreostatics, pyrazolone derivatives. Iodine deficiency leads to increased synthesis of TSH, which causes hyperplasia of the gland parenchyma. Local changes in the struma are determined by its size. In young patients, a diffuse struma initially develops, which is replaced by a nodular form. Possible compression of the larynx with the development of stridor. In addition, acromegaly, Usher syndrome (autosomal dominant (A/D) transmission; double upper lip; blepharochalasis; pigment spots), Pendred syndrome (autosomal recessive transmission - A/R) occur with struma syndrome; internal hearing loss; violation of vestibular functions; minimal hypothyroidism), Refetow-Wind-Groot syndrome (congenital general resistance to thyroid hormone; congenital deafness leading to muteness; “bird face”, “chicken breast” and “wing-shaped shoulder blades”; tachycardia without signs of hyperthyroidism), Siple syndromes and Wermer [3]. Sometimes thyroid dysfunction syndrome in seriously ill people is discussed as a separate condition. The syndrome is defined as an abnormality of thyroid function tests in clinically euthyroid patients with severe nonthyroidal systemic diseases (anorexia nervosa, chronic renal failure, liver cirrhosis, major surgery, major burns, sepsis, etc.). T3 level is reduced. Hyperthyroidism - increased release of thyroxine and/or triiodothyronine with the development of the corresponding clinical picture. In most cases, hyperthyroidism occurs as Graves' disease. Less commonly, the cause is autonomy of the thyroid gland. In other situations, hyperthyroidism is caused by thyroiditis with increased gland function, exogenous overdose of hormones, iodine, the presence of thyrotropin-secreting tumors of the anterior pituitary gland, pituitary gland uncontrolled by the hypothalamus hypersecretion of thyroid-stimulating hormone, malignant tumors of the thyroid gland, ectopic secretion of the hormone in the ovarian struma. In addition, hyperthyroidism syndrome occurs (except for the already described thyroiditis) Troel-Jannet syndrome (diffuse hyperostosis of the integumentary bones of the skull with acromegaloid hyperthyroidism), fibrous dysplasia and McCune-Albright syndrome. Graves' disease is an autoimmune thyropathy with hyperthyroidism, struma, ophthalmopathy, pretibial myxedema and tachycardia. In the Graves form of hyperthyroidism, most patients have immunoglobulins (antibodies to thyroglobulin and microsomal antigen), the disease has autoimmune features and is genetically determined (A/R). The gland, as a rule, is enlarged in size; it is often possible to palpate the sound of flowing blood (Fig. 1). The clinical picture is quite variable, but in classic cases it does not cause difficulties. Patients are irritable, emotionally labile, speech is rapid, feverish, sweating is increased, patients cannot tolerate heat. Appetite is increased, despite sufficient calorie supply. Tachycardia, arrhythmia, small-amplitude tremor of the fingers, moist warm thin soft skin, and dyspigmentation are noted. Thin hair, increased hair loss, brittle nails, frequent stool disorders, peripheral swelling. Skin swelling similar to periorbital swelling develops on the legs (infiltrative dermopathy). The maximum severity is on the anterolateral surfaces, the skin is hyperemic, reminiscent of an orange peel. In this place, patients feel itching. The skin soon turns from pink to brown. Pretibial infiltrative dermopathy rarely appears without association with ophthalmopathy. Like ophthalmopathy, it can appear years before manifestations of thyrotoxicosis or years after its apparent completion. With a long course of uncompensated hyperthyroidism, acropachydermia is possible = hypertrophy of the skin, tubular bones and their ends with the formation of “drumsticks”. Adolescent girls develop hypo-, oligo- and amenorrhea, and boys develop decreased potency and gynecomastia. Eye symptoms are extremely important. Exophthalmos and eyelid edema combined with symptoms of “setting sun” (stripe of tunica albuginea above the iris = retraction of the upper eyelid), Stellwag (rare blinking), Mobius (weakness of convergence), Graefe (lag of the upper eyelid when looking down), Dalrympa (retraction of the upper eyelid as a result of spasm of M. Levator palpebrae when looking forward), Kocher (lag in eye movement from the upper eyelid when looking up quickly),

Rosenbach (trembling of the upper eyelid with eyes closed), increased tear formation. Persistent tachycardia with possible atrial fibrillation leads to heart failure. Neuromuscular syndrome manifests itself as thyrotoxic myopathy, predominantly in the proximal muscles, although a mosaic combination of damage to distal and proximal muscles is possible. Creatine kinase was not elevated in all cases. Masked hyperthyroidism usually occurs with cardiac symptoms. Typical are heart failure that responds poorly to cardiac glycosides, atrial fibrillation with a slow ventricular response, other paroxysmal or latent arrhythmias, and cardiomegaly. Gastrointestinal disorders include loss of appetite, weight loss, hepatomegaly, constipation. Possible depletion of psychomotor activity, “senile” dementia, osteoporosis, bone pain, and ease of fractures. The diagnosis is difficult in low-symptomatic forms, which can occur under the guise of apathy, general weakness, weight loss or cardiomyopathies with atrial fibrillation until the development of heart failure. In such situations, the thyroid gland is usually not enlarged or slightly enlarged. The diagnosis of hyperthyroidism is confirmed by identifying an increased level of free thyroxine and a decreased TSG level. In 4% of patients with hyperthyroidism, an isolated increase in triiodothyronine levels is detected. In all types of thyrotoxicosis, a test with thyroid-releasing factor is usually negative, i.e. not defined an increase in TSH levels in response to the introduction of a synthetic releasing factor, although positive results do not exclude hyperfunction of the thyroid gland. An increase in the basal level of TSH (normal = 1-6 mU/l) is determined only for pituitary tumors that secrete thyroid-stimulating hormone. Autonomy of the thyroid gland develops with uninodular or multinodular autonomous adenomas (Plummer's disease). Their endocrine activity does not depend on the pituitary influence. In the clinical picture All nuances from eu- to hyperthyroidism are possible. Autonomy of the thyroid gland can develop in patients with struma nodosa and increased iodine intake (amiodarone, X-ray contrast agents). For the first In such cases, cardiac symptoms usually appear.

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