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Thyrotoxicosis and Atrial Fibrillation

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Thyrotoxicosis is a clinical syndrome caused by the effect of excess thyroid hormones on the body. The term “hyperthyroidism” is also used to describe this syndrome - a state of hyperproduction of thyroid hormones by the thyroid gland, both pathological and physiological (for example, during pregnancy). The frequency of detection of hyperthyroidism in patients with atrial fibrillation (AF), according to various authors, ranges from 5 to 63% and depends on many factors: the age and gender of the patients, the initial state of the cardiovascular system and thyroid gland, the treatment, and also on the severity iodine deficiency in the region where the subjects live.
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In a study by JCForfar et al. Among patients with idiopathic AF, 13% were diagnosed with thyrooxycosis. According to the results of the Canadian Registry of Patients with AF among persons with newly diagnosed AF or atrial flutter (AF), a decrease in the level of thyroid-stimulating hormone (TSH) was detected in 5.5% [8]. According to Italian researchers, among patients with paroxysmal AF who did not have electrocardiographic and echocardiographic signs of any cardiomyopathy, low TSH was detected in 63% [128]. Moldavian authors found thyrotoxicosis in 26.8% of patients with paroxysmal and persistent AF who did not have severe somatic diseases and were not taking amiodarone [4]. According to our data, in newly diagnosed AF and AFL, low TSH levels were detected in 18% of patients [14], and among all hospitalized patients with AF and AFL - in 9.5% [23]. In turn, in patients with thyrotoxicosis, AF is the second most common cardiac complication after sinus tachycardia and is detected in 10-15% of patients compared to 4% in the general population. As in the general population, the prevalence of AF in thyrotoxicosis increases with age, being less than 5% in people under 60 years of age and 25-40% in people over 60 years of age [27]. Compared with the general population, the presence of thyrotoxicosis in patients is associated with increased cardiovascular and cerebrovascular mortality. Moreover, the main cause of unfavorable outcomes in these patients is AF, as it leads to the development of heart failure and cerebral embolism.

Although ADKrahn et al. believe that clinically significant dysfunction of the thyroid gland in patients with newly diagnosed AF is rarely diagnosed and routine testing of TSH levels is advisable only in patients with additional symptoms of thyrotoxicosis; guidelines for the management of patients with AF [5] recommend mandatory testing of TSH levels in all individuals with newly diagnosed AF, as well as when tachysystole persists against the background of antiarrhythmic treatment or with early resumption of arrhythmia after cardioversion. The need to study TSH levels in all patients with recently developed and otherwise unexplained AF is explained by the possibility of restoring euthyroidism and, accordingly, sinus rhythm (SR) in patients with identified thyrotoxicosis [26].

1. THYROID FUNCTION AND HEART

1.1. Basic provisions

The thyroid gland synthesizes two hormones that directly control the activity of the cardiovascular system - thyroxine (T4) and triiodothyronine (T3). The necessary substrate for their synthesis is iodine. After entering the body, inorganic iodine selectively accumulates in the thyroid gland, where, through the process of oxidation, molecular iodine is formed, which becomes an integral part of T4 and T3 (the numbers indicate the number of iodine atoms in the molecule). T4 and T3 are associated with the protein thyroglobulin, which is the matrix for their synthesis and subsequent storage in the thyroid gland. Although T4 is the main product of the thyroid gland and is itself capable of exerting a number of effects through its own receptors in target cells, T3 is biologically active, which is formed from T4 under the action of enzymes that remove iodine (deiodinases). The biological activity of T3 is 5 times higher than that of T4. More than 99% of T4 and T3 circulating in the blood are in protein-bound form and, as needed, quickly turn into the hormonally active free form (free T4 and free T3), which penetrates the target cells. The production of thyroid hormones is controlled by TSH of the pituitary gland, which, in turn, is controlled by the hypothalamus, which produces thyrotropin-releasing hormone. Regulation of the secretion of TSH and thyroliberin is carried out using a negative feedback mechanism and is closely related to the level of T4 and T3 in the blood: with a decrease in the level of T4 and T3, the secretion of TSH and thyroliberin quickly increases and the concentration of thyroid hormones is restored. Thyroid hormones are necessary for the normal development of all organs and systems, primarily the nervous, cardiovascular and musculoskeletal systems. The main function of thyroid hormones at the intrauterine stage of development is tissue differentiation. Under their influence, the development of brain structures, the formation and maintenance of intelligence throughout life, and the growth of bones occur. The influence of thyroid hormones on the cardiovascular system is manifested by positive inotropic, chronotropic, dromotropic and bathmotropic effects, leading to increased and increased heart rate, improved conduction of excitation through the myocardium and increased excitability of the heart muscle, as well as a decrease in systemic peripheral resistance.

1.2. Laboratory assessment of thyroid function

Laboratory diagnosis of thyroid function disorders is based on determining the levels of TSH, free T4 and free T3. The main method for assessing thyroid function is to determine the TSH level. Each person has a genetically determined normal level of free T4, and minimal stable changes in this level, even those within the range of accepted normal values, are perceived by the pituitary gland as a deviation from the norm, leading to an exponential change in the level of TSH: when free T4 is reduced by half, TSH secretion increases in 100 times. That is why determining the TSH level is the most sensitive way to detect minimal functional disorders of the thyroid gland. Considering that the value of total T4 and T3 depends to a greater extent on the concentration of binding proteins than on the degree of dysfunction of the thyroid gland, in order to determine the function of the thyroid gland, the determination of free rather than total T4 and T3 has diagnostic significance. The use of total T4 and total T3 levels in the blood in routine clinical practice is considered diagnostically irrational and economically ineffective. Currently, the study of TSH levels is carried out using 3rd generation reagents with a sensitivity of 0.01-0.02 mU/l, which allows not only to identify the fact of a decrease in TSH levels, but also to distinguish the degree of this decrease. According to modern concepts, for adults the normal limits for TSH range from 0.4 to 4.0 mU/l (or μU/ml). If the lower limit of the TSH norm depends on the sensitivity of the reagents used, then the upper limit is the result of the consensus of experts. This is explained by the fact that the optimal values of the upper limit of normal TSH in relation to survival in different categories of individuals differ. Thus, according to a Norwegian population-based study in initially healthy women, the risk of death from coronary heart disease (CHD) was lower with TSH from 0.5 to 1.4 mU/L than with a higher level [28]. In older people, on the contrary, better survival was observed among those whose TSH was above the upper limit of normal.

The range of normal values of thyroid hormones for free T4 is from 9.0 to 23.0 pmol/l (0.7-1.8 ng/dl), for free T3 - from 3.5 to 7.7 pmol/l (0.2 0.5 ng/dl). Depending on the level of TSH, free T4 and free T3, three states of functional activity of the thyroid gland are distinguished: euthyroidism, hypothyroidism and thyrotoxicosis. Hypothyroidism and thyrotoxicosis are divided into manifest and subclinical. Euthyroidism is characterized by normal levels of TSH and thyroid hormones. Hypothyroidism is classified into primary (caused by the destruction or lack of functionally active thyroid tissue), as well as secondary and tertiary (or hypothalamic-pituitary, central). To verify the diagnosis of hypothyroidism, it is enough to examine the level of TSH and free T4, since the level of free T3 in hypothyroidism remains normal for a long time and decreases only in severe forms. The diagnosis of manifest hypothyroidism is established with an increased level of TSH and a decreased level of free T4,subclinical hypothyroidism - with elevated TSH and normal levels of free T4. Secondary and tertiary hypothyroidism is characterized by normal or decreased TSH and decreased free T4. When identifying laboratory signs of hypothyroidism, it is advisable to conduct an additional study of antithyroid antibodies to thyroid peroxidase (AT-TPO), which makes it possible to establish the cause of hypothyroidism and predict the transition of subclinical hypothyroidism to manifest: an increase in AT-TPO indicates autoimmune thyroiditis (AIT) as the cause of primary hypothyroidism and is a precursor. speaker of the transition of subclinical hypothyroidism to the manifest form. To confirm the autoimmune nature of the disease, it is sufficient to study only TPO Ab, and simultaneous study of TPO Ab and antibodies to thyroglobulin is considered economically inappropriate. Thyrotoxicosis can be endogenous (caused by a primary thyroid disease) or exogenous (caused by excessive administration of thyroid hormones from the outside). Thyrotoxicosis, accompanied by increased secretion of thyroid hormones, during isotope scanning of the thyroid gland is characterized by increased uptake of radiopharmaceuticals (RP), and caused by the destruction of thyrocytes and exogenous - by its low uptake. Laboratory diagnosis of thyrotoxicosis involves the study of three hormones: TSH, free T4 and free T3. Manifest thyrotoxicosis is characterized by a decrease in TSH, an increase in free T4 and an increased or normal level of free T3. Subclinical thyrotoxicosis is diagnosed in cases where the TSH level is reduced, and the levels of free T4 and free T3 are within normal limits. The first step in diagnosing thyrotoxicosis is to determine the TSH level: if it is normal, thyrotoxicosis is excluded. If a reduced TSH level is detected, the free T4 level is additionally examined. The combination of a decreased TSH level and an increased free T4 indicates manifest thyrotoxicosis; with a decrease in TSH and a normal level of free T4, it is necessary to examine the level of free T3, since in approximately 2-3% of patients thyrotoxicosis manifests as isolated T3 thyrotoxicosis. The procedure for carrying out the listed hormonal studies in practice turns out to be quite arbitrary, and in some centers it is customary to immediately determine all three hormones, which leads to an increase in the cost of the examination, while others have adopted a stepwise principle, which, while lengthening the time before diagnosis, is still considered more rational [15]. Correct organization of work to study thyroid function involves a single blood draw from the patient followed by storage of the serum in the laboratory for 2-7 days, so that if TSH deviates from the norm, the level of thyroid hormones can be consistently determined from the same serum. Which makes it possible to establish the cause of hypothyroidism and predict the transition of subclinical hypothyroidism to manifest: an increase in AT-TPO indicates autoimmune thyroiditis (AIT) as the cause of primary hypothyroidism and is a predictor of the transition of subclinical hypothyroidism to the manifest form. To confirm the autoimmune nature of the disease, it is sufficient to study only TPO Ab, and simultaneous study of TPO Ab and antibodies to thyroglobulin is considered economically inappropriate. Thyrotoxicosis can be endogenous (caused by a primary thyroid disease) or exogenous (caused by excessive administration of thyroid hormones from the outside). Thyrotoxicosis, accompanied by increased secretion of thyroid hormones, during isotope scanning of the thyroid gland is characterized by increased uptake of radiopharmaceuticals (RP), and caused by the destruction of thyrocytes and

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1.3. Variants of the clinical course of thyrotoxicosis

The main clinical symptoms of thyrotoxicosis are: nervousness, emotional lability (detected in 99%); general tremor, hand tremor (85-97%); hot and damp skin (97%); tachycardia (89-95%); increased fatigue (65-88%); weight loss (85%); low-grade fever; shortness of breath (45-75%); brittle nails, hair loss; eye discomfort (54%); diarrhea (33%); menstrual irregularities (22%); splenomegaly (10%), thyrotoxic hepatosis; angina pectoris (3-5%). A life-threatening complication of thyrotoxicosis is a thyrotoxic crisis, characterized by the maximum severity of symptoms of thyrotoxicosis, the development of adrenal insufficiency, and severe systemic decompensation. Clinically, it is manifested by severe mental anxiety, motor hyperactivity, alternating apathy and disorientation, severe hyperthermia, abdominal pain, vomiting, cardialgia, and suffocation.

Cardiac symptoms(palpitations, decreased exercise tolerance, shortness of breath, angina-like chest pain, peripheral edema, congestive heart failure) are often predominant in the clinical picture of thyrotoxicosis. Patients with a long history of thyrotoxicosis and persistent tachysystolic AF may develop symptoms of both left ventricular (shortness of breath on exertion, orthopnea, paroxysmal nocturnal dyspnea) and right ventricular failure (distension of the jugular veins, liver enlargement, edema of the lower extremities).

A long history of thyrotoxicosis leads to left ventricular hypertrophy and systolic arterial hypertension (AH) with normal or reduced diastolic pressure. Thyrotoxicosis often contributes to the development of pulmonary hypertension, as increased cardiac output leads to overload of the right heart and stretching of the tricuspid valve ring. In elderly people and with multinodular toxic goiter (MTG) in regions of iodine deficiency, T3 thyrotoxicosis is diagnosed in some cases, clinically manifested by attacks of tachycardia or AF, emotional lability and depression.

Since thyrotoxicosis leads to an increase in myocardial oxygen consumption, it can cause cardiac decompensation in elderly people with underlying cardiovascular disease (CVD). In elderly people, thyrotoxicosis has few symptoms or is asymptomatic. Asymptomatic ("apathetic") thyrotoxicosis is observed in 15% of patients over 70 years of age. The elderly are characterized by a low incidence of goiter (50% of cases) and a predominance of cardiac pathology (congestive heart failure and AF) in the clinical picture. In 15% of elderly people, thyrotoxicosis clinically manifests as new-onset AF. Among the clinical symptoms of thyrotoxicosis in the elderly, the most frequently recorded are apathy, tachycardia and weight loss.

Thyrotoxicosis is associated with decreased vagal activity and decreased heart rate variability, which may persist despite restoration of euthyroidism. ECG allows us to identify patients with hyperthyroidism who have a high risk of developing AF: increased duration and dispersion of the P wave are significant predictors of AF for both subclinical and manifest thyrotoxicosis [20].

2. NON-THYROID CAUSES OF CHANGES IN HORMONE LEVELS

Euthyroid sick syndrome is a change in the level of thyroid hormones in the blood in individuals without thyroid disease. Other designations for this syndrome: pseudothyroid dysfunction, euthyroid weakness syndrome, nonthyroidal illness syndrome. Various acute and chronic CVDs (heart surgery, acute myocardial infarction, heart failure), as well as any severe somatic diseases (liver disease, severe infections, uremia, ketoacidosis, surgical interventions, prolonged fasting, extensive burns) can alter the metabolism of thyroid hormones, leading to a decrease in the level of total (more often) and free (less often) T3. Levels of T4 and TSH may either remain normal or slightly decrease or increase. These laboratory changes are often interpreted as adaptive, aimed at conserving calories and protein in the body.

The mechanism for reducing T3 levels is multifactorial and is partly related to the suppression of peripheral 5-monodeiodination of T4 in the liver. In patients with congestive heart failure, the degree of decrease in free T3 is proportional to the severity of the NYHA functional class of heart failure. A decrease in T3 levels is detected in almost 30% of patients with chronic heart failure (CHF), both in those taking and not taking amiodarone. A low level of fT3 in patients with CVD is an independent predictor of both cardiovascular and overall mortality. It remains not entirely clear whether severe somatic pathology can cause "tissue hypothyroidism," which has its own pathological significance. Works have been published that demonstrate improvement in cardiac output and peripheral vascular resistance with intravenous administration of T3 to patients with CHF [68] after coronary artery bypass surgery [8], but data on improving the prognosis for these patients are not yet available. Currently, the

generally accepted position is that the administration of thyroid hormones in euthyroid pathology syndrome is not indicated.

In acute somatic diseases, the TSH level may become subnormal for a short time and gradually recover during the recovery stage, sometimes increasing to 10-20 mU/l. The limits of normal TSH values in this case expand to 0.05-10.0 mU/l and are regarded as euthyroidism or minimal dysfunction of the thyroid gland, which can be confirmed or excluded by repeated laboratory monitoring 2-3 months after recovery.

In patients with AF, it is advisable to retest TSH, fT4 and fT3 after 2 weeks. If a dysfunction of the thyroid gland is nevertheless suspected, then a TPO-AT study may be useful for differential diagnosis between autoimmune thyroid disease and non-thyroid causes. It is very difficult to diagnose subclinical hypothyroidism in somatically decompensated patients during hospitalization due to the frequent increase in TSH levels in these individuals, which is caused by the disease itself, and not by a decrease in thyroid function. The diagnosis of true hypothyroidism in patients with severe somatic diseases is characterized by a combination of low free T4 and high TSH (>20 mU/l). It is believed that with normal fT4 levels, TSH fluctuations in the range between 0.02-20.0 mU/l (subclinical hyper- or hypothyroidism) are unlikely to affect the outcome of somatic disease, and control of tests can be delayed for 2-3 months until recovery. In older adults, the interpretation of laboratory results presents significant difficulties due to age-related changes in thyroid function and frequent changes due to non-thyroid diseases and medications [29].

Thus, in one prospective study, during a screening study of TSH and fT4 levels in all patients hospitalized for 3 months in a geriatric clinic, euthyroid pathology syndrome was identified in 17.9% of individuals. Many drugs cause changes in the metabolism of thyroid hormones. Thus, a decrease in TSH levels occurs when using dopamine, dobutamine and dobutrex, as well as glucocorticosteroids (GCS), which directly suppress the secretion of TSH by the pituitary gland; when taking beta-blockers, an increase in T4 levels may occur due to a slowdown in the conversion of T4 to T3; non-steroidal anti-inflammatory drugs, furosemide, heparin and sulfonylurea derivatives displace T3 and T4 from binding to proteins, leading to a decrease in total and an increase in free T3 and T4; Amiodarone has a complex effect on thyroid function (see below). Thus, identifying hypothyroidism and thyrotoxicosis in patients with AF and concomitant chronic or acute somatic diseases can present significant difficulties, therefore, laboratory signs of thyroid dysfunction in the absence of a clinical picture of hypothyroidism or thyrotoxicosis first of all require rechecking the results.

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