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FEATURES OF ATRIAL FIBRILLATION DEVELOPMENT IN PATIENTS WITH THYROTOXICOSIS

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Annotation: *The article presents a review of the literature on the development of atrial fibrillation in patients with thyrotoxicosis, which is one of the most common endocrine diseases, second only to diabetes mellitus.*

The most common cause of thyrotoxicosis is Graves ' disease and functional autonomy of the thyroid node. The authors analyze the literature data on the cardiac effects of thyrotoxicosis, the features of cardiac remodeling in hyperthyroidism, the frequency of atrial fibrillation in thyrotoxicosis depending on age, as well as the possibilities of restoring sinus rhythm in a combination of these pathologies. Special attention is paid to the effect of subclinical thyrotoxicosis on the heart, which is understood as a violation of thyroid function, characterized by a low serum concentration of thyrotropin, normal values of free thyroxine and free triiodothyronine. Subclinical thyrotoxicosis can also cause cardiac remodeling and diastolic dysfunction. The prevalence of thyrotoxicosis in the elderly is higher in areas of iodine deficiency, which is relevant for our country due to the large area of iodine deficiency. In elderly patients, the cardiac effects of thyrotoxicosis predominate in the clinical picture, which makes it difficult to diagnose endocrine pathology, and correction of thyrotoxicosis is critical for successful heart rhythm control. The article also discusses the problem of thyrotoxic cardiomyopathy caused by the toxic effect of an excess of thyroid hormones: features of this heart lesion, factors affecting its formation, clinical significance and contribution to the development of rhythm disorders. The greatest significance is the development of atrial fibrillation against the background of thyrotoxicosis in patients of the older age group, which is already burdened with various cardiovascular diseases. Atrial fibrillation is the most common cardiac arrhythmia in thyrotoxicosis. The formation of arrhythmia in hyperthyroidism is based on the simultaneous existence of frequent focal impulses and circular movements of the excitation wave-the re-entry mechanism. Successful treatment of atrial fibrillation in patients with thyrotoxicosis is possible only when euthyroidismis achieved. In most cases, when thyrotoxicosis is stopped, spontaneous recovery of the sinus rhythm is noted. The chances of restoring sinus rhythm are lower in elderly patients with concomitant organic myocardial pathology or long-term atrial fibrillation. It should be remembered that even a radically cured thyrotoxicosis leads to a deterioration in the life prognosis.

Keywords: *thyrotoxicosis, atrial fibrillation, thyrotoxic cardiomyopathy, left ventricular hypertrophy*

Introduction

Thyrotoxicosis (TSH) is a syndrome caused by an excessive content of thyroid hormones in the blood and their toxic effect on various organs and tissues. The prevalence of TTD varies from region to region, which primarily depends on the level of iodine intake, sampling criteria in studies, the influence of factors such as age, gender, genetic and environmental characteristics, as well as the technical characteristics of methods

for determining the level of thyroid hormones. TTZ ranks second in the structure of endocrine pathology (after diabetes mellitus). The most common cause of TTD is Graves ' disease, functional autonomy of the thyroid node in patients with nodular / multi-nodular goiter, and less often-thyroiditis. The prevalence of TTD among women is 0.5-2%, and among men it is 10 times less [1-6]. Pathological changes in the cardiovascular system are already mentioned in the first descriptions of TSH. Graves R. A. himself described goiter and ocular symptoms as secondary to heart damage. Moebius P .J. suggested the determining role of the thyroid gland in the genesis of cardiovascular damage in Graves ' disease. Kraus R. in 1899 .he first introduced the term "thyrotoxic heart", and G. F. Lang used this term for the first time in Russia in 1936 [7].

Cardiac effects of thyrotoxicosis The cardiac effects of TSH are most dangerous for the elderly, but they often dominate the clinic. The prevalence of TTZ among the elderly depends on the degree of iodine deficiency, on average from 0.4 to 2% [2, 8]. In areas of iodine deficiency, which include almost the entire Russian country, the prevalence of TTD in the elderly is higher due to a higher incidence of toxic autonomy [9-11], and reaches 15% in people over 75 years of age [12]. The concept of thyrotoxic cardiomyopathy (CMC) has been used in the internal medicine clinic since 1995, after the introduction of the WHO classification of cardiomyopathies. Epidemiological and diagnostic data there are no criteria. Thyrotoxic CMP is a complex of disorders of the cardiovascular system caused by the toxic effect of excess thyroid hormones. This condition often not only comes to the fore in the clinical picture, but also determines the course and outcome of the disease, especially if the patient has a concomitant cardiac pathology. The severity of thyrotoxic CM depends on the age, duration and severity of TTD, and concomitant diseases. The prevalence of thyrotoxic CMT: left ventricular hypertrophy, left atrial dilatation, atrial fibrillation, pulmonary hypertension, and heart failure is high in young and middle-aged patients with thyrotoxicosis. Thyrotoxic CMP is formed under the influence of both unmodifiable (gender, age, genesis of thyrotoxicosis) and modifiable factors (systolic arterial hypertension, duration of thyrotoxicosis, level of thyroid hormones and thyroid-stimulating hormone). The greatest contribution to the formation of thyrotoxic CMP is made by the duration of TTZ and the age of patients, the contribution of other factors to the development of individual manifestations of CMP in thyrotoxicosis is variable. According to available data [13-16], in clinical TTD, changes in the heart develop under the influence of: a) direct trophoprivate action of thyroid hormones on the myocardium by activating the expression of a number of genes, in particular, contractile proteins; b) through activation of the sympathetic nervous system by increasing the expression of the β1-adrenoreceptor gene and increasing the density of β1-adrenoreceptors on cardiomyocytes; c) through the renin-angiotensin system by activating the expression of angiotensin 2 and renin genes both in the cardiomyocyte and in peripheral tissues (kidney, vascular wall). Cardiac hyperfunction in TSH inevitably leads to hypertrophy with a 30-50% increase in myocardial mass [16,25,25]. There are few studies that study the features of left ventricular remodeling in clinical and subclinical thyrotoxicosis in the available literature. Meanwhile, in the literature there are data on the detection of both normal geometry and myocardial hypertrophy in patients with TTZ – eccentric and concentric. The reason for the existing disagreements is not fully clear, probably the nature of the geometry of the left ventricle is influenced by the age and gender of patients, the duration of thyrotoxicosis, and concomitant diseases. In most cases (about 70%), the normal geometry of the left ventricle is preserved. Eccentric left ventricular hypertrophy (LVH) develops with a long, recurrent course of the disease, and concentric LVH develops in people over 45 years of age [17, 18]. There is evidence that concentric LVH is the most unfavorable prognostic factor. It is associated with the highest incidence of adverse cardiovascular events and mortality [17,23,23]. It can be assumed that the increase in cardiovascular mortality in patients with a history of thyrotoxicosis, both clinical and subclinical, noted by many authors, is a consequence, among other things, of changes in the geometry of the left ventricle in these patients. With a prolonged course of TSH, protein synthesis in the myocardium decreases, dystrophy and cardiosclerosis develop,

resulting in congestive heart failure. With development and progressionin heart failure, the blood flow rate decreases, minute and systolic blood volumes decrease, and peripheral vascular resistance increases. Hyperdynamia is replaced by hypodynamia characterized by an increase in the period of tension due to the phases of asynchronous and isosynchronous contraction with a shortened period of expulsion [7,11,11]. Thus, the nature of left ventricular myocardial remodeling in TTD is determined by the influence of a number of factors, among which the leading value is the severity and duration of TTD, age, and gender. It is impossible to exclude the influence of other factors that are not reliably established, for example, iodine availability.

Thyrotoxicosis and atrial fibrillation The most common cardiac arrhythmia in TSH should be considered atrial fibrillation. Occurrence of atrial fibrillation in patients with TTD accordingto various authors [1, 9, 15] ranges from 5 to 22%. According to the Endocrinological Research Center, the incidence of "thyrotoxic heart damage" was 6.1%, and atrial fibrillation was 4.8%. The prevalence of atrial fibrillation in the general adult population is 1-2%. With the development of TTD, the frequency of detection of atrial fibrillation increases and reaches 2.86% in men and 1.36% in women, although women suffer from TTD 10 times more often [22]. Factors affecting the probability of atrial fibrillation in TSH include male gender, age, and the presenceof concomitant cardiovascular diseases [9, 19]. Thus, the incidence of AF in TTD is less than 5% in people under 60 years of age, and 25-40% in people over 60 years of age [15, 16]. In the Frost L study. et al. (2004) the incidence of atrial fibrillation in persons with TTD aged 20-29 years left 0.3%, and reached 19% at the age of 80-89 years [9]. Dystrophic atrial lesions play a role in the origin of atrial fibrillation in TSH. Electrocardiographic signs of right atrial hypertrophy, often combined with dilation, were registered in 30.5% of patients with thyrotoxicosis, left atrial hypertrophy-in 13.7%, and hypertrophy of both atria-in 11.8% of patients. Atrial dilation, their increased excitability in combination with the ease of occurrence of functional blockages due to heterogeneity of various parts of the myocardium.the re-entry mechanism is based on [25]. In TS, there is a functional heterogeneity of various parts of the myocardium. Any additional impact that increases this heterogeneity can cause complete discoordination of the activity of various parts of the heart muscle, which is manifested by various arrhythmias. A long-standing discussion about the underlying mechanism of atrial fibrillation — re-entry or focal pulse generation-has led to the conclusion that frequent focal impulses and circular movements of the excitation wave simultaneously exist [19]. In recent years, there have been studies devoted to the study of the effect of subclinical TTZ on the heart. The term "subclinical TSH" refers to a thyroid disorder characterized by a low serum concentration of thyrotropin (Despite its almost asymptomatic course, it can adversely affect the cardiovascular system, causing an increase in myocardial mass and wall thickness of the left ventricle, the development of diastolic dysfunction and dilatation of the left atrium. In experimental studies in animal models, thyroxine suppression of thyroid-stimulating hormone led to the development of concentric LVH. However, it remains unclear whether subclinical TTZ is capable of causing LVH in humans, and what type of left ventricular myocardial remodeling is characteristic of it. It is known that subclinical TTZ has the greatest negative impact on patients of older age groups, but the age threshold from which this effect begins has also not been established. In addition, it is known that age is an independent determinant of LVH. It seems that the contribution of the sympathetic nervous system in subclinical TTD is significantly less significant, since an increase in heart rate and other manifestations of sympathoadrenal activity are observed only in a part of patients. It can be assumed that the increase in the activity of the renin-angiotensin system in these patients becomes dominant, which leads to the predominance of concentric forms of remodeling. Probably, the leading factors determining the development of LVH in subclinical TTD are still the age of patients and the duration of the disease. Long-term maintenance of thyroid-stimulating hormone suppression in patients receiving therapy for clinical TSH also contributes to the development of concentric forms of remodeling. Reducing the risk of cardiac changes in TSH can be achieved by maintaining persistent euthyroidism during conservative treatment, including normalization of

thyroid-stimulating hormone, adequate correction of systolic arterial hypertension, including the use of ACE inhibitors, and earlier radical treatment of patients with factors that increase the risk of developing thyrotoxic CM. If euthyroidism is achieved in a timely manner, rhythm disturbances, in particular, atrial fibrillation, will also be reversible. According to Nakazama et al., 62% of patients spontaneously recovered their sinus rhythm 8-10 weeks after reaching euthyroidism[20]. The chances of restoring sinus rhythm are reduced in elderly patients with concomitant organic myocardial pathology or long-term fibrillation atrial fibrillation (more than 4 months) [15,1-8]. However, in young patients without concomitant heart diseases, an attempt should be made to restore and maintain sinus rhythm 3 months afterthe achievement of persistent euthyroidism [15, 22]. Patients with severe progressive TTD may develop a permanent form of atrial fibrillation after a series of paroxysms – a consequence of persistent sinus node dysfunction. Even radically treated TTZ leads to a deterioration in the life prognosis. Thus, mortality from cardiovascular diseases, arterial hypertension, valvular malformations, rhythm disorders, heart failure, and, to a lesser extent, from ischemic heart disease increased in this cohort by 1.2 times, and from cerebrovascular disease- by 1.4 times compared to the general population [1-1, 17]. A population – based study [25] of long-term TTD outcomes was conducted in Great Britain and Wales from 1950 to 1989, during which the "endpoints" - mortality, myocardial infarction, and stroke-were evaluated in patients with TTD who received radioiodine therapy . The follow-up period was 105 person – years, and the number of patients with TTD was 7209. The number of patients who died significantly exceeded the expected mortality calculated using the standard mortality rate for the general population. Among the leading causes of death were cardiac and cerebral accidents. Similar data were obtained by other researchers: it was shown that the development of TTZ in people over 50 years of age significantly worsens the prognosis of concomitant diseases such as arterial hypertension, cerebral and coronary atherosclerosis. The reason for the increase in mortality was the development of persistent changes in the cardiovascular systemduring the TTZ period [7, 20, 25,].

ConclusionThus, the greatest attention from the clinician requires TTZ in elderly patients, for whom its timely diagnosis is extremely important. It should be remembered that 15% of elderly patients with TTZ clinically manifest first-time atrial fibrillation. In the cohort of elderly patients, only decompensation of cardiovascular diseases, depression, and rhythm disorders can be clinical manifestations of TSH. On the other hand, it is advisable to monitor thyroid hormones in all patients with first-time atrial fibrillation, as well as with difficult heart rate control in the case of cordarone use and with early resumption of arrhythmia after cardioversion.

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