Original article:

Thyroid status in the combination of atrial fibrillation with subclinical thyrotoxicosis

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Abstract

Objective: To study the factors in the development of atrial fibrillation in patients with subclinical hyperthyroidism on the background of angina pectoris as a result of a comprehensive analysis of the electrocardiographic and echocardiographic indicators of the heart, thyroid volume, thyroid hormone levels and lipid profile. ***Materials and Methods:*** The risk factors for atrial fibrillation (AF) were studied in patients with subclinical hyperthyroidism (SH) and angina pectoris. It was revealed that the starting factor for the occurrence of AF in patients with subclinical hyperthyroidism is a shift in the reference values of thyroid hormones. It was established that the total depression of the ST segment reflects the degree of coronary reserve in patients with AF on the background of SH. ***Results and Discussion***: It has been shown that in patients with subclinical hyperthyroidism, there is a normal level of total cholesterol and LDLP, a low level of triglycerides and atherogenicity, and a high level of HDLP. When combined with subclinical hyperthyroidism and paroxysmal AF, there is an increase in the level of total cholesterol, LDLP, TG, atherogenicity and decreased HDLP. ***Conclusion***: It has been proven that diastolic dysfunction of the left ventricle of the first type is formed on the background of SH.

Keywords: atrial fibrillation, subclinical hyperthyroidism

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Introduction

Atrial fibrillation (AF) is the most common type of tachyarrhythmia[1]. The prevalence of AF is from 2 to 4% in people aged 50 to 75 years, and in patients older than 75, 85, and 90 years, its prevalence is 5, 14, and 27%, respectively[2].

In numerous large studies conducted in persons older than 55-60 years, it was found that the prevalence of patients with TSH levels less than 0.1 mMED / l ranges from 0.7 to 12.4%[3]. It was also found that the prevalence of people with TSH levels less than 0.1 mMED / l increases to 15.0% in patients over 70 years old in areas with iodine deficiency[4]. Many researchers believe that the risk of atrial fibrillation in patients with subclinical hyperthyroidism is 2.5 times higher[5]. It was also established that with the combination of subclinical hyperthyroidism with IHD, the frequency of occurrence of AF increases 4-6 times.

Analysis of the literature shows that the diagnosis of the treatment of atrial fibrillation is most fully described in the manifest form of hyperthyroidism[6]. At the same time, a comprehensive assessment of electrocardiographic and echocardiographic parameters of the heart, the thyroid status and lipid metabolism in patients with paroxysmal atrial fibrillation with a combination of subclinical hyperthyroidism was not carried out with angina

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To study the factors in the development of atrial fibrillation in patients with subclinical hyperthyroidism on the background of angina pectoris as a result of a comprehensive analysis of the electrocardiographic and echocardiographic indicators of the heart, thyroid volume, thyroid hormone levels and lipid profile.

Material and research methods.

As a result of immunochemical screening of thyroid function, in 826 patients with AF, 139 patients (62 men and 77 women) aged from 58 to 67 years old (average age 62.7 ± 2.4 years) were isolated and divided into 4 groups. The 1st group included 34 patients with asymptomatic paroxysms of AF against the background of a combination of stable angina pectoris of the II functional class with subclinical hyperthyroidism. The 2nd group included 32 patients with symptomatic paroxysmal AF on the background of a combination of stable angina pectoris of II functional class with subclinical hyperthyroidism. In the 3rd group, 38 people are combined with asymptomatic paroxysms of AF against the background of stable angina pectoris II functional class. The 4th group consisted of 35 patients with symptomatic paroxysmal AF on the background of stable angina pectoris II functional class.

The 1st control group included 33 patients (average age - 60.8 ± 2.2 years) with subclinical hyperthyroidism. The 2nd control group included 30 healthy individuals (mean age - 60.4 ± 2.5 years).

All patients included in the study were recorded initial ECG in 12 standard leads, Holter ECG Monitoring (HM), echocardiography (EchoCG), transesophageal electrophysiological study (TeES) of the heart, ultrasound of the thyroid gland, determination of the TH level in the blood were performed.

Ultrasound examination (UE) of the thyroid gland was performed using an Aloka ultrasound scanner (Japan) equipped with a 7 MHz linear sensor. The basal level of TSH, triiodothyronine (T3f), and thyroxin (T4f) were studied by an enzyme immunoassay using standard kits.

EchoCG was performed for all patients in accordance with the standards of the American Association for Echocardiography using the Sanos-100CF apparatus (Hewlett-Packard, USA) with sinus rhythm (6). The indicators of the systolic function of the left ventricle were determined: indices of the final systolic and terminal diastolic volumes (EDV, ESV), impact index (II), ejection fraction (EF), anterior - posterior size of the left atrium (LA). To assess the diastolic function of the left ventricle, the following transmitral blood flow parameters were calculated: the maximum rate of early diastolic filling (peak E), the maximum filling rate of the left ventricle during atrial systole (peak A), the ratio of these velocities E / A.

For the diagnosis of symptomatic and asymptomatic atrial fibrillation, HM was performed. Detection of paroxysm of AF for more than 30 seconds, accompanied by clinical symptoms, was considered as a symptomatic relapse during HM. The presence of paroxysm of AF for longer than 30 seconds and without clinical symptoms was considered as asymptomatic relapse during HM. If patients had both symptomatic and asymptomatic relapses of AF, these relapses were treated as symptomatic.

TeES conducted the conventional protocol using the electrophysiological complex "Astrocard" in the modes of competitive, programmed and frequent stimulation at a tape speed of 25, 50, 100 mm / s and signal amplification of 1 mV - 10, 20 mm. The duration of the RR, PQ, QT intervals, the width of the QRS complex, the recovery time of the sinus node function (SNRT) and its corrected value (CSNRT), the Venkebach point, the effective refractory period of the atrioventricular (ERPAV) compound and the left atrium (ERPLA) were determined.

The diagnosis of angina pectoris II functional class exhibited when discomfort behind the sternum occurred as a result of fast walking or fast climbing stairs, after eating or in the cold, or in windy weather, or under the influence of emotional stress, or in the first few hours after getting out of bed; while walking a distance of more than 200 m or two quarters on flat terrain or while climbing a ladder for more than one span at a normal pace under normal conditions.

As a diagnosis of coronary reserve in angina pectoris of the II functional class, we used the method of increasing transesophageal heart stimulation (TeS). For this stimulation was performed with a frequency of 15-20 bpm exceeding the patient's heart rate. Every 2 min the stimulation frequency was increased by 20 bpm until the appearance of a horizontal depression of the ST segment on the ECG is not less than 0.2 mV. The maximum frequency of stimulation in all groups was 160 bpm. During CPF and at the end of each stage, ECG was recorded in 12 leads and evaluated with a total ST segment depression in branches aVF, III, V3, V4 (mV).

Statistical processing of the research results was carried out on a personal computer using the Statistica for Windows software package from Stat-Soft Inc using parametric and non-parametric criteria.

Ethical approval:
This research was approved by Ethics Committee of Penza State University, Penza, Russian Federation, Russia

Results of the study

Comparative evaluation of the history of AF and its characteristics showed that the duration of arrhythmia, the number of paroxysmal AF, and the duration of paroxysm of AF in groups depends on the clinical manifestations of arrhythmia and the layering of subclinical hyperthyroidism (Table 1.)

Table 1. Characteristics of AF in subclinical hyperthyroidism (M ± m)

Indicators	1st group (ASAFAPSH) n=34 n/% 1	2nd group (SAFAPSH) n=32 n/% 2	3rd group (ASAFAP) n=38 n/% 3	4th group (SAFAP) n=35 n/% 4
Duration of AF, years	6,4±0,34	5,1±0,30 p_{1-2}=0,0096	5,8±0,32 p_{1-3}=0,2306 p_{2-3}=0,1207	4,5±0,26 p_{1-4}<0,001 p_{2-4}=0,1384 p_{3-4}=0,0061
Numbers of paroxysms, per year	18,1±1,0	14,3±0,8 p_{1-2}=0,0074	15,4±0,9 p_{1-3}=0,0421 p_{2-3}=0,3836	13,7±0,8 p_{1-4}=0,0027 p_{2-4}=0,5748 p_{3-4}=0,1655
Duration of AF (min)	24,5±1,33	18,3±1,02 p_{1-2}<0,001	22,2±1,26 p_{1-3}=0,2124 p_{2-3}=0,0167	16,2±0,98 p_{1-4}<0,001 p_{2-4}=0,1441 p_{3-4}<0,001
VE AF (bpm)	146,5±7,9	152,6±8,6 p_{1-2}=0,5753	145,3±8,3 p_{1-3}=0,7805 p_{2-3}=0,5307	151,4±8,9 p_{1-4}=0,6299 p_{2-4}=0,7846 p_{3-4}=0,585
HR SR, bpm	63,6±3,4	81,8±4,6 p_{1-2}=0,0059	62,3±3,6 p_{1-3}=0,7033 p_{2-3}=0,0042	78,3±4,6 p_{1-4}=0,011 p_{2-4}=0,5685 p_{3-4}=0,0034

Abbreviation: ASAFAPSH – asymptomatic atrial fibrillation with angina pectoris in subclinical hyperthyroidism, SAFAPSH - symptomatic atrial fibrillation with angina pectoris in subclinical hyperthyroidism, ASAFAP - asymptomatic atrial fibrillation with angina pectoris , SAFAP - symptomatic atrial fibrillation with angina pectoris, VE AF – ventricular extrasystoles during AF episode, HR SR – heart rate in sinus rhythm.

Compared with symptomatic paroxysmal AF in patients with asymptomatic over, the experience of arrhythmia was 22.4% more (p=0.0061), the number of paroxysms per year - by 11.0% (p=0.1655), the duration of AF - by 27.0% (p <0.001). The combination of subclinical hyperthyroidism with AF was accompanied by an increase in the experience of arrhythmia, the number of paroxysms and duration of AF by 20.0% (p = 0.0096), 21.1% (p = 0.0074) and 25.3% (p <0.001), respectively.

Heart rate with sinus rhythm in the 2nd group was more often than in the 1st by 28.6% (p = 0.0059), and in the 4th, than in the 3rd by 25.7% (p = 0.0034). Also ERP LA in the 2nd group was more than in the 1st by 27.4% (p = 0.0332), and in the 4th, than in the 3rd by 25.1% (p = 0.0402).

The mechanisms and conditions under which the development of AF in symptomatic and asymptomatic paroxysms of tachycardia and when combined with subclinical hyperthyroidism are currently not well understood. Therefore, the objective of this study was a comprehensive study of these dependencies with a combination of cardiac and thyroid pathology.

The current state of thyroidology does not allow for an optimal approach to solving the problem of etiological diagnosis of subclinical hyperthyroidism[7]. Therefore, our findings are of great theoretical and practical importance.

Analyzing the etiology of subclinical hyperthyroidism in the main and control groups , we found that its main causes are diffuse toxic goiter (31.3-33.3%), multinodular toxic goiter (21.9-27.3%) toxic adenoma of the thyroid gland (18.2–20.6%), less often benign tumors of the thyroid gland (3.0–5.9%), exogenous administration of thyroid hormones (2.9–6.1%), rare diseases of the thyroid gland (2 ,9-3.1%). In addition, in a number of patients we were unable to establish the cause of subclinical hyperthyroidism, therefore, we identified idiopathic causes of subclinical hyperthyroidism (9.1-15.6%).

Studies have shown that in patients with asymptomatic, symptomatic AF and healthy individuals, the volume of the thyroid gland, the level of TSH and thyroid hormones (T4f, T3f) do not differ significantly (p. 0.05) (Table 2). At the same time, in a detailed analysis of the distribution of patients with asymptomatic AF, symptomatic AF and healthy individuals, depending on the upper and lower reference ranges of T4f and T3f , we found significant differences (Table 2).

Table 2. The distribution of patients with AF, subclinical hyperthyroidism and healthy individuals, depending on the upper and lower reference range of free T4 and T3.

Indicators	ASAFAPSH	SAFAPSH	ASAFAP	SAFAP	SH	HP
	n/%	n/%	n/%	n/%	n/%	n/%
	1	2	3	4	5	6
T4f ur, pm/l	10/26,3	25/71,4	8/23,5	26/81,3	22/67	16/53,3
T_4f lr, pm/l	28/73,7	10/28,6	26/76,5	6/18,8	11/33	14/46,7
		χ^2_{1-2}=14,9 p_{1-2}= 0,0001	χ^2_{1-3}=0,07 p_{1-3}= 0,7852	χ^2_{1-4}=21,0 p_{1-4}=0,00001	χ^2_{1-5}=11,6 p_{1-5}=0,0007	χ^2_{1-6}=5,2 p_{1-6}=0,0228
			χ^2_{2-3}=15,9 p_{2-3}= 0,0001	χ^2_{2-4}=0,89 p_{2-4}=0,346	χ^2_{2-5}=0,18 p_{2-5}=0,6710	χ^2_{2-6}=2,3 p_{2-6}=0,1318
				χ^2_{3-4}=22,0 p_{3-4}= 0,00001	χ^2_{3-5}=12,6 p_{3-5}=0,0004	χ^2_{3-6}=6,04 p_{3-6}=0,0140
					χ^2_{4-5}=1,79 p_{4-5}=1,181	χ^2_{4-6}=5,52 p_{4-6}=0,0188
						χ^2_{5-6}=1,2 p_{5-6}= 0,28
T3f ur, pm/l	10/26,3	27/77,1	7/20,6	25/78,1	20/60,6	18/60,0
T3f lr, pm/l	28/73,7	8/22,9	27/79,4	7/21,9	13/39,4	12/40,0
		χ^2_{1-2}=18,8 p_{1-2}= 0,00001	χ^2_{1-3}=0,33 p_{1-3}=0,5678	χ^2_{1-4}=18,7 p_{1-4}=0,00001	χ^2_{1-5}=8,5 p_{1-5}=0,0035	χ^2_{1-6}=7,9 p_{1-6}=0,0051
			χ^2_{2-3}=22,1 p_{2-3}=0,00001	χ^2_{2-4}=0,01 p_{2-4}=9233	χ^2_{2-5}=2,18 p_{2-5}=0,140	χ^2_{2-6}=2,23 p_{2-6}=0,1355
				χ^2_{3-4}=21,9 p_{3-4}=0,00001	χ^2_{3-5}=11,2 p_{3-5}=0,0008	χ^2_{3-6}=10,4 p_{3-6}=0,0013
					χ^2_{4-5}=2,34 p_{4-5}=0,126	χ^2_{4-6}=2,39 p_{4-6}=0,1219
						χ^2_{5-6}=0,001 p_{5-6}= 0,9608

Abbreviations: ASAFAPSH – asymptomatic atrial fibrillation with angina pectoris in subclinical hyperthyroidism, SAFAPSH - symptomatic atrial fibrillation with angina pectoris in subclinical hyperthyroidism, ASAFAP - asymptomatic atrial fibrillation with angina pectoris, SAFAP - symptomatic atrial fibrillation with angina pectoris, SH – subclinical hyperthyroidism, HP – healthy peoples.

The study showed, that in the studied groups, depending on the level of T4f and T3f, the patients were on different reference ranges. Healthy individuals were balanced at the level of the upper and lower range. Compared with healthy individuals , patients with subclinical hyperthyroidism were concentrated in the side of the upper reference range ($\chi 2$ =1.2, p=0.28), when combined asymptomatic AF with subclinical hyperthyroidism - in the bottom (χ =5.2, p=0,0228), and symptomatic AF with subclinical hyperthyroidism - at the top ($\chi 2$=2.3, p=0.1318), asymptomatic AF - at the bottom ($\chi 2$=6.04, p =0.0144), symptomatic AF - in top ($\chi 2$=5.52, p=0.0188).

Currently, there is no consensus about the mechanisms of the emergence of asymptomatic and symptomatic AF[8]. Based on the data obtained, it is obvious that a shift in the level of T4f and T3f to the upper and lower range of the reference value may affect the clinical manifestations of AF. Obviously, when the levels of T4f and T3f are shifted to the upper range of the reference value, symptomatic AF occurs, and to the lower one - asymptomatic AF.

In recent years, great importance is attached to the issue of assessing the degree of coronary reserve in various comorbitic states[9]. The literature on this issue is controversial[10]. However, with the introduction of non-invasive research methods into clinical practice, the possibility of diagnosing the degree of coronary reserve has increased significantly[11].

The data we obtained on the results of the assessment of the degree of coronary reserve are presented in Table 3.

Table 3. Total ST segment depression with angina pectoris II FC

Indicators	1st group (ASAFAPSH) n=34	2nd group (SAFAPSH) n=32	3rd group (ASAFHF) n=38	4th group (SAFAP) n=35
	1	2	3	4
∑□↓ ST at aVF, III, V3, V4 in patients with AP IIFC	0,44±0,056	0,42±0,051	0,38±0,044 p_{1-3}=0,038	0,35±0,036 p_{2-4}=0,045
ERP LA, ms	210,7±15,0	268,5±20,1 p_{1-2}=0,0332	212,5±15,1 p_{1-3}=0,7839 p_{2-3}=0,0400	265,9±18,9 p_{1-4}=0,0345 p_{2-4}=0,7785 p_{3-4}=0,0402

Abbreviations: ASAFAPSH – asymptomatic atrial fibrillation with angina pectoris in subclinical hyperthyroidism, SAFAPSH - symptomatic atrial fibrillation with angina pectoris in subclinical hyperthyroidism, ASAFAP - asymptomatic atrial fibrillation with angina pectoris, SAFAP - symptomatic atrial fibrillation with angina pectoris, ERP LA – effective refractory period of the left atrium.

We found that the total segment depression is maximal in patients with asymptomatic paroxysms of AF against the background of a combination of angina pectoris voltage II FC with subclinical hyperthyroidism. It was also found that the total depression of the ST segment with asymptomatic paroxysmal AF is greater than with symptomatic. It should also be noted that the layering of subclinical hyperthyroidism on symptomatic and asymptomatic paroxysms of AF significantly ($p < 0.05$) increases the total depression of the ST segment. As can be seen from the obtained data, the total depression of the ST segment is unreliable ($p > 0.05$) more in case of subclinical hyperthyroidism.

The performed correlation analysis showed that there is an inverse relationship between the total depression of the ST segment and ERP LA ($p < 0.05$).

Based on the above data, it can be concluded that TeES with an assessment of the total depression of the ST segment and ERP LA allows evaluating the functional class of angina pectoris and the dispersion of refractory periods of the atria in patients with AF on the background of subclinical hyperthyroidism.

According to a number of researchers, lipid metabolism in subclinical hyperthyroidism has a complex pathogenesis[12]. The work on this issue has been fragmented and carried out in various laboratories, differing from each other in a number of lipid profile parameters[13].

The results of our studies in patients with subclinical hyperthyroidism, when combined subclinical hyperthyroidism with symptomatic paroxysmal AF and subclinical hyperthyroidism with asymptomatic paroxysmal AF are presented in Table 4.

Table 4. Lipid profile in patients with subclinical hyperthyroidism, when combined subclinical hyperthyroidism with symptomatic paroxysmal AF and subclinical hyperthyroidism with asymptomatic paroxysmal AF (M ± m)

Indicators	Subclinical hypethyroidism	Subclinical hyperthyroidism with symptomatic AF	Subclinical hyperthyroidism with asymptomatic AF
	1	2	3
Total cholesterol, mmol/l	4,9±0,288	5,3±0,301 p_{1-2}=0,347	5,6±0,303 p_{1-3}=0,0854 p_{2-3}=0,486
LDLP, mmol/l	2,8±0,167	3,5±0,200 p_{1-2}=0,006	3,6±0,193 p_{1-3}<0,001 p_{2-3}=0,708
HDLP, mmol/l	1,5±0,090	0,98±0,056 p_{1-2}<0,001	0,92±0,049 p_{1-3}<0,001 p_{2-3}=0,435
TG, mmol/p	1,2±0,071	2,4±0,136 p_{1-2}<0,001	2,6±0,140 p_{1-3}<0,001 p_{2-3}=0,327
Atherogenicity	2,2±0,131	4,4±0,250 p_{1-2}<0,001	5,09±0,275 p_{1-3}<0,001 p_{2-3}=0,070

From the above data, it follows that in patients with AF paroxysms, on the background of subclinical hyperthyroidism, there is a normal level of total cholesterol and LDLP, a low level of triglycerides and atherogenicity and a high level of HDLP. As can be seen from the Table 4 with a combination of subclinical hyperthyroidism and symptomatic AF, there is an increase in the level of total cholesterol by 8.4% (p=3470), LDLP - by 24.7% (p = 0.002), TG - by 98.4% (p<0.001), atherogenic coefficient - by 98.7% (p <0.001) and HDLP decrease by 35.5% (p<0.001), and then with the combination of subclinical hyperthyroidism with asymptomatic AF by 14.5% (p=0.0854) , 27.2% (p<0.001), 114.9% (p<0.001), 129.3% (p<0.001), 39.5% (p<0.001), respectively.

The mechanisms and conditions under which AF arises are currently not well understood[14, 15]. Echocardiography is used to identify structural changes in the myocardium. However, echocardiography in individuals with AF against the background of a combination of cardiac and thyroid pathology was performed in single studies.

Analysis of hemodynamic parameters, the diameter of the common carotid artery (CCA) in groups of patients before inclusion in the study is presented in Table 5.

Abbreviations: LA –left atrium, EDS – end diastolic size, ESS- end systolic size, EF – ejection fraction, LV – left ventricular, E - early diastolic filling, A – systole of atrium, IVRT – isovolumic relaxation time, DT – left ventricular contractility.

It was revealed that the volume of the left atrium, IVRT in patients with AF and subclinical hyperthyroidism was significantly greater (p <0.05) compared with healthy individuals. Peak E, E/A in patients with AF and subclinical hyperthyroidism was significantly lower than in healthy ones.

Result and Discussion

1. Compared with symptomatic paroxysmal AF in patients with asymptomatic languor over, the experience of arrhythmia is 22.4% more (p = 0.0061), the number of seizures per year is 11.0% (p = 0.1655), the duration of AF - by 27.0% (p <0.001). The combination of subclinical hyperthyroidism with AF was accompanied by

Table 5. Analysis of hemodynamic parameters, the diameter of the common carotid artery (CCA)

Indicators	ASAFAPSH	SAFAPSH	ASAFAP	SAFAP	SH	HP
	1	2	3	4	5	6
LA volume, ml/m^2	29,5±1,52	27,7±1,56	27,3±1,54	26,5±1,50	24,6±1,45 p1-5=0,0197	23,5±1,47 p1-6=0,0091, p2-6=0,0471
EDS, mm	52,1±2,78	51,6±2,91	51,1±2,86	50,5±3,03	50,2±2,95	49,6±2,84
ESS, mm	35,2±1,90	34,9±2,1	34,6±1,95	34,2±2,05	33,9±1,97	33,5±1,92
EF, %	60,2±3,31	59,6±3,40	60,2±3,48	60,8±3,59	61,4±3,63	62,1±3,75
Septal thickness, mm	11,1±0,61	10,7±0,65	10,9±0,70	10,8±0,64	10,7±0,60	10,6±0,63
Posterior wall thickness, mm	12,2±0,71	12,1±0,67	11,9±0,69	11,8±0,66	11,7±0,71	11,6±0,71
LV mass, g/m^2	94,5±5,10	93,5±5,32	92,6±5,24	91,7±5,38	90,8±5,34	89,9±5,40
E, cm/s	80,4±4,4	82,9±4,7	85,4±4,88	88,1±5,21	93,7±5,43	156,2±9,46 p1-6, 2-6, 3-6, 4-6, 5-6<0,001
A, cm/s	85,9±4,6	87,2±4,9	89,3±5,08	89,4±5,26	93,1±5,34	93,6±5,71
E/A	0,936±0,0504	0,951±0,0533	0,956±0,056	0,9855±0,073	1,006±0,0628	1,669±0,1004 p1-6, 2-6, 3-6, 4-6, 5-6<0,001
IVRT	97,2±5,31	96,3±4,80	95,3±5,37	94,4±5,56	93,4±5,47	69,2±4,20 p1-6, 2-6, 3-6, 4-6, 5-6<0,001
DT	217,6±11,7	215,5±12,20	213,3±12,11	211,2±12,48	209,1±12,35	199,2±12,10
Diameter CCA, mm	5,8±0,16	5,6±0,12	5,5±0,14	5,4±0,17	5,3±0,13	5,2±0,14

an increase in the experience of arrhythmia, the number of seizures and the duration of AF by 20.0% (p = 0.0096), 21.1% (p = 0.0074) and 25.3% (p <0.001), respectively.

2. The main causes of subclinical hyperthyroidism are diffuse toxic goiter (31.3-33.3%), multinodular toxic goiter (21.9-27.3%), toxic thyroid adenoma (18.2-20.6%), less often - benign tumors of the thyroid gland (3.0-5.9%), exogenous administration of thyroid hormones (2.9-6.1%), rare thyroid diseases (2.9-3.1%).
3. In healthy individuals, there was a balanced distribution of thyroid hormones at the level of the upper and lower ranges. Compared with healthy individuals, patients with subclinical hyperthyroidism were concentrated in the side of the upper reference range ($\chi 2$ = 1.2, p = 0.28), when combined asymptomatic AF with subclinical hyperthyroidism - in the bottom ($\chi 2$ = 5.2, p = 0, 0228), and symptomatic AF with subclinical hyperthyroidism - in the upper ($\chi 2$ = 2.3, p = 0.1318), asymptomatic AF - in the lower ($\chi 2$ = 6.04, p = 0.0140), symptomatic AF - in the upper ($\chi 2$ = 5.52, p = 0.0188).
4. Total segment depression is maximal in patients with asymptomatic paroxysms of AF against the background of a combination of angina pectoris tension II FC with subclinical hyperthyroidism. The total depression of the ST segment with asymptomatic paroxysmal AF is greater than with symptomatic. The layering of subclinical hyperthyroidism on symptomatic and asymptomatic paroxysms of AF significantly (p <0.05) increases the total depression of the ST segment. There is an inverse relationship between total depression of the ST segment and ERP LA (p <0.05).
5. In patients with subclinical hyperthyroidism,

there is a normal level of total cholesterol and LDLP, a low level of triglycerides and atherogenicity, and a high level of HDLP. In subclinical hyperthyroidism combination with symptomatic AF is an increase in total cholesterol at 8,4% (p = 3,470), LDLP - by 24,7% (p = 0,002), TG - by 98,4% (p <0,001), atherogenic coefficient - by 98.7% (p <0.001) and HDLP decrease by 35.5% (p <0.001), and then with the combination of subclinical hyperthyroidism with asymptomatic AF by 14.5% (p = 0.0854), 27,2% (p <0.001), 114.9% (p<0.001), 129.3% (p <0.001), 39.5% (p <0.001), respectively.

6. The volume of the left atrium, IVRT in patients with AF and subclinical hyperthyroidism is significantly greater (p<0.05) compared with healthy individuals. Peak E, E/A in patients with AF and subclinical hyperthyroidism was significantly lower than in healthy ones.

Conclusions

1. The starting factor for the onset of AF in patients with subclinical hyperthyroidism is: in symptomatic paroxysms, the reference values of thyroid hormones are shifted to the upper level, and asymptomatic - to the lower level.
2. The total depression of the ST segment reflects the degree of coronary reserve in patients with AF on the background of SH. There is an inverse correlation between the amounts of depression of the ST segment and ERP LA.
3. In patients with subclinical hyperthyroidism, there is a normal level of total cholesterol and LDLP, a low level of triglycerides and atherogenicity and a high level of HDLP. When combined subclinical hyperthyroidism with symptomatic AF, there is an increase in the level of total cholesterol by 8.4% (p =3470), LDLP - by 24.7% (p = 0.002), TG - by 98.4% (p <0.001), atherogenicity - by 98.7% (p <0.001) and HDLP decrease by 35.5% (p <0.001), and then when combined with subclinical hyperthyroidism and asymptomatic AF by 14.5% (p = 0.0854), 27, 2% (p<0.001), 114.9% (p <0.001), 129.3% (p <0.001), 39.5% (p <0.001), respectively.
4. Against the background of SH, diastolic LA dysfunction of the first type is formed - peak E, E/A and an increase in the volume of the left atrium and IVRT.

Conflict of interest: There is no conflict of interest

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