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FEATURES OF CENTRAL HEMODYNAMICS IN PATIENTS WITH DIFFUSE TOXIC GOITER

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Abstract

The aim of the study isto study the features of central hemodynamics in patients with diffuse toxic goiter.

Material and methods of research. 32 people were examined, including 22 women, 10 men, average age49.2±1.62years (for women -47±1.76years, for men -42±2.6years)

Research methods – general clinical, biochemical (bilirubin, direct, indirect, ALT, AST, PTI, coagulogram, CRP), hormonal (TSH, free thyroxine, antibodies to thyroid peroxidase, to thyroglobulin and thyrocyte receptors, prolactin in the blood),anthropometric calculations (height (cm), weight (kg), BMI (kg/m2), waist circumference, waist circumference) and instrumental: ECG, echo-ECG, ultrasound of the thyroid gland, internal organs, chest X-ray

Research results. When analyzing echocardiography data in patients with thyrotoxicosis syndromevisiblethat in the general group of patients, a reliable decrease in the indicators of end-diastolic volume and end-diastolic volume and a reliable decrease in the ratio of peaks E and A in relation to the control were observed.

An analysis of echocardiography parameters in patients with thyrotoxicosis depending on the clinical form showed that in the overt form, the EDV and ESV values, peak E, and the E/A ratio are significantly reduced. In the subclinical form, the IVS and peak A values are significantly increased, and a significant decrease in ESV is observed.

Conclusions.Echocardiographic signs of hemodynamic disturbances in thyrotoxicosis can be detected even before the manifestation of clinical symptoms of thyrotoxic heart disease.

Keywords: Graves' disease, hemodynamics, degree.

Introduction

It is known that excess thyroid hormones negatively affect the cardiovascular system.[1-4].It accelerates the heartbeat, strengthens cardiac contractions, increases the volume of ejected blood, and increases total cardiac output. At the same time, it reduces vascular resistance and shortens the heart muscle relaxation time. Long-term exposure to such conditions can aggravate existing cardiac problems and lead to the development of thyrotoxic cardiomyopathy.[5-8].This condition is characterized by thickening of the left ventricular wall, enlargement of its cavities, and impaired relaxation, which can ultimately lead to heart failure and arrhythmias such as atrial fibrillation.[9-12].Although these abnormalities are often reversible with treatment of excess hormones, they sometimes persist even after thyroid function has returned to normal.[13-15].Because thyrotoxic cardiomyopathy is a rare cause of heart failure, its prevalence, course, and prognosis are poorly understood.[16-18].Additionally, patients with excess thyroid hormone often have other heart conditions, making it difficult to understand the connection between hyperthyroidism and heart problems.[18].

Heart failure (HF) develops in 5.4% of patients with Graves' disease and most commonly presents as HF with preserved ejection fraction (HFpEF). Independent risk factors for HF with reduced ejection fraction (HFrEF) include atrial fibrillation (AF) and elevated thyroid-stimulating hormone receptor antibodies (TRAb), while for HFpEF, they include COPD, older age, overt hyperthyroidism, high body mass index (BMI), and hypertension. Elevated thyroid hormone levels were risk factors for both types of HF, but HFpEF occurred with moderate elevations in free thyroxine (FT4) levels. Both types were associated with a higher risk of cardiovascular hospitalization, and HFpEF was additionally associated with a higher incidence of ventricular tachycardia/ventricular fibrillation (VT/VF) and all-cause mortality.[6].

Their key finding showed that heart failure developed in 5.4% of patients with Graves' disease. However, the factors predisposing to heart failure with reduced ejection fraction (HFrEF) and preserved ejection fraction (HFpEF) differed. For HFrEF, the key risk factors were atrial fibrillation (AF) and high levels of TSH receptor antibodies (TRAb). Meanwhile, for HFpEF, COPD, older age, severe hyperthyroidism, elevated BMI, and hypertension were more significant. Most patients with newly diagnosed HFrEF associated with Graves' disease experienced an improvement in ejection fraction after treatment of the underlying disease. However, less than half of patients with HFpEF reported improvement in heart failure symptoms and diastolic function, as measured by echocardiography. Patients with additional cardiovascular risk factors were more likely to have persistent left ventricular dysfunction or heart failure. Both types of heart failure (HFpEF and HFrEF) resulted in increased cardiovascular hospitalizations. However, only HFrEF was associated with increased all-cause mortality and the risk of ventricular arrhythmias (VT/VF).[6].

All of the above formed the basis for this study.

The aim of the study is to investigate the features of central hemodynamics in patients with diffuse toxic goiter.

Material and methods of research

32 people were examined, including 22 women, 10 men, average age49.2±1.62years (for women -47±1.76years, for men -42±2.6years)

The control group consisted of20 healthy individuals (10 women and 10 men) whose hormonal parameters were used to assess the reliability of the results obtained.

*Inclusion criteria:*diffuse toxic goiter, men, women
*Exclusion criteria:*The study did not include patients with carbohydrate metabolism disorders, ischemic heart disease, heart defects, idiopathic cardiomyopathy, as well as patients who had suffered a cerebrovascular accident or other causes of hyperthyroidism.

Research methods

General clinical, biochemical (bilirubin, direct, indirect, ALT, AST, PTI, coagulogram, CRP), hormonal (TSH, free thyroxine, antibodies to thyroid peroxidase, to thyroglobulin and thyrocyte receptors, prolactin in the blood),anthropometric calculations (height (cm), weight (kg), BMI (kg/m2), waist circumference, waist circumference) and instrumental: ECG, echo-ECG, ultrasound of the thyroid gland, internal organs, chest X-ray

Echocardiography (EchoCG) was performed on a Sonoline GGOS (Siemens) device; a 3.15 MHz sensor was used to scan the structures of the heart in standard echocardiographic positions. (City Clinical Hospital No. 1, Andijan).

The assessment of the cavity sizes, the interventricular septum (IVS) thickness, and the posterior wall (PW) thickness was carried out according to the standard methodology of the American Association of Echocardiography (ASE, 2005). The following indices of the structural and functional state of the LV were determined: end-diastolic dimension (EDD), end-diastolic volume (EDV), end-systolic dimension (ESD), end-systolic volume (ESV), stroke volume (SV), LV posterior wall thickness in diastole (LVPTD), and interventricular septum thickness in diastole (IVSTD).

The mass of the left ventricular myocardium was calculated using the formula[1].

LV ML=1.04 ({TMZHP+LV TZSL=LV EDR}3)-13.6g, where LVM is the left ventricular mass, 1.04 is the cardiac muscle density coefficient, IVST is the interventricular septum thickness, LPV is the left ventricular posterior wall thickness, and LVEDD is the left ventricular end-diastolic dimension. The left ventricular mass index (LVMI) was calculated as the ratio LVM/BSA, where BSA is the body surface area determined by the formula of D. Dubois (1975) (LVH was defined as LVMI values greater than 96 g/m2 in women and greater than 115 g/m2 in men, according to the 2005 ASE recommendation.

To assess the geometric remodeling of the LV, remodeling indices such as the relative wall thickness (RWT) and the sphericity index (SI) of the LV were used. RWT of the LV was calculated using the formula:

TVR = (TZSLZhD + TMZhPD) / LV EDR

Values >0.45 were taken as an increase in the LV TSR. To distinguish the types of LV geometry, the classification of G. Ganau of 1992 was used, according to which 4 variants of LV remodeling are distinguished: normal geometry (NG) - no LV hypertrophy (LVH), TSR<0.45; eccentric hypertrophy (EH) - there is LVH, TSR<0.45, concentric remodeling (CR) - no LVH, TSR>0.45; concentric hypertrophy (CH) - there is LVH, TSR>0.45.[1, 2].

Statistical softwareMicrosoft Excel and STATISTICA_6 was used for statistical analysis, and $p < 0.05$ was considered a significant difference. Quantitative data with normal distribution were expressed as mean and standard deviation (M ± SD).

Research results. Distribution of patients by age groupsgiven in Table 1.

Table 1 Distribution of patients by age groups

Age group	Men (n=10)		Women (n=22)	
	number of observations	%	number of observations	%
Up to 20 years old	-	-	-	%
21-30 years old	-	-	5	22.7
31-40 years old	8	80.0	15	68.2
41-50 years old	2	20.0	2	9.09
51-60 years old	-	-	-	-
Over 61 years old	-	-	-	-

Note: n is the number of patients examined.

During examination of the patients, the following changes were observed: the apex beat became more pronounced, heart sounds increased in intensity, and the second heart sound over the pulmonary artery became more distinct. Auscultation also revealed a functional systolic murmur at the apex, at the fifth auscultation point, and over the pulmonary artery. The cardiac muscle expanded toward the left.

A study of changes in heart rate (HR) and blood pressure (BP) was conducted in patients with overt (manifest) and latent (subclinical) thyrotoxicosis, comparing them with the parameters of the control group. According to the data in Table 2, in patients with overt thyrotoxicosis, the average HR was 105.95±1.69 beats per minute. Systolic blood pressure reached 149.1±2.12 mmHg, and diastolic – 87.3±21.03 mmHg. These values were statistically significantly higher than in the control group. In patients with subclinical thyrotoxicosis, HR averaged 103.3±3.22 beats per minute, and BP – 144.7±4.12 mmHg. (systolic) and 86.9±2.41 mmHg (diastolic). These indicators were also significantly different from those in the control group.

Table 2 Hemodynamic parameters in patients with varying degrees of thyrotoxicosis

	Control Group (n=20)	Subclinical thyrotoxicosis (n= 10)	Thyrotoxicosis Manifest (n=22)
Heart rate bpm	72.6±1.03	103.3±3.22*	105.95±1.69*
Systolic blood pressure mmHg	113.25±2.33	144.7±4.12*	149.1±2.12*
Diastolic blood pressure (BP) mmHg	68.25±1.63	86.9±2.41	87.3±21.03

Note: n – number of examined patients;
*-reliability in relation to control (*p<0.0005)

When analyzing echocardiography data in patients with thyrotoxicosis syndromevisiblethat in the general group of patients there is a significant decrease in the indicators of end-diastolic volume and end-diastolic volume and a significant decrease in the ratio of peaks E and A in relation to the control (Table 3)

Table 3 Changes in echocardiography parameters in patients with thyrotoxicosis.

Indicators	Control group (n=20)	Total number of patients (n=32)
LP	2.63±0.12	3.23±0.11*
MZhP	0.82±0.02	0.96±0.04*
KDR	4.52±0.13	4.76±0.09*
ZSLZh	0.99±0.05	1.01±0.03*
KSR	2.70±0.11	3.00±0.09*
KDO	120.5±2.57	107.1±5.21*
KSO	52.5±1.33	34.04±2.61*
FV	67±0.86	66.43±1.39
PIC E	0.99±0.06	0.57±0.05*
PIK A	0.40±0.005	0.87±0.07*
E/A	2.48±0.17	0.65±0.03*

Note: n – number of patients examined; * – presence of reliability in relation to the control (*p<0.05), LV TWP, mm — left ventricular posterior wall thickness; LV EDD, mm — left ventricular end-diastolic dimension; LV RWT — left ventricular relative wall thickness; LARV, mm — left atrial volume; LVMI — LV mass index; IVST — interventricular septum thickness; *p< 0.05.

According to two-dimensional echocardiography, no cases of impaired local contractility of the LV were noted.

The LV EDV index in patients with thyrotoxicosis (107.1±5.21*) was significantly lower than in the control group (120.5±2.57 ml; p<0.05). The average LV ESV in patients was also significantly lower (34.04±2.61*) than in the control group (52.5±1.33 ml; p<0.05). Indexed LV EDV and ESV in patients with thyrotoxicosis syndrome had statistically significant differences compared to the control group data (p<0.05).

An analysis of echocardiography parameters in patients with thyrotoxicosis depending on the clinical form revealed that in the overt form, the EDV and ESV values, peak E, and the E/A ratio are significantly reduced. In the subclinical form, the IVS and peak A values are significantly increased, and a significant decrease in ESV is noted. It is evident that the maximum changes are characteristic of the overt form of thyrotoxicosis, although signs reflecting ventricular afterload are already observed in subclinical thyrotoxicosis (Table 4).

Table 4. Indicatorsleft ventriclein those examined, depending on the form of thyrotoxicosis

Indicators	Manifest hyperthyroidism (n=22)	Subclinical hyperthyroidism (n=10)	Control group (n=20)
LP	3.09±0.13*	3.11±0.24	2.63±0.12
MZhP	0.88±0.05*	1.05±0.09*	0.82±0.02
KDR	4.77±0.09*	4.69±0.35*	4.52±0.13
ZSLZh	1.00±0.01*	0.99±0.08	0.99±0.05
KSR	3.02±0.33	2.89±0.18	2.70±0.11
KDO	104.1±4.85*	99.5±26.6*	120.5±2.57
KSO	35.2±2.45*	30.2±11.9*	52.5±1.33
FV	65.6±1.47	69.8±3.75	67±0.87
PIC E	0.58±0.05*	0.59±0.02*	0.99±0.06
PIK A	0.84±0.07*	0.80±0.06*	0.40±0.005
E/A	0.60±0.03*	0.72±0.06*	2.48±0.17

Note: n – number of examined patients;
*-reliability in relation to control (*p<0.05)

Left ventricular mass, left ventricular mass index, and relative left ventricular wall thickness were calculated using the formulas of Devereux R. and Reicheck (1977) and D. Dubois (1975). The values were significantly reduced compared to the control group (Table 5).

Table 5 Left ventricular parameters in the examined patient groups

Indicator	General group (n=32)	Control group (n=20)
Myocardial mass, g	414.2±12.6*	110±8.9
Myocardial mass index	65.5±4.5*	68.9±9.8
OTC	0.40±0.01 *	0.43±0.02
BDO, ml	107.8±3.55*	123.4±2.15
KSO, ml	37.7±1.38*	55.9±2.31
FV,%	64.7 ±0.79*	61.6±1.43

In our study, which analyzed myocardial diastolic function, we identified left ventricular diastolic dysfunction in patients with subclinical and overt thyrotoxicosis. This dysfunction was characterized by an increase in the peak A velocity, a decrease in the E/A ratio, an increase in the left ventricular early diastolic filling time (DTE), and an increase in the left ventricular isovolumetric relaxation time (IVRT). Our data correlate with the results of studies by Biondi et al., Mercuro O. et al., and Smit J. et al. [3-5], who also studied myocardial diastolic function.

Conclusions

Echocardiographic signs of hemodynamic disturbances in DTG can be detected even before the manifestation of clinical symptoms of thyrotoxic heart disease.

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