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DISRUPTION OF CATECHOLAMINE METABOLISM IN WOMEN WITH MICROVASCULAR ANGINA

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Abstract:
This scientific article examines the relationship between the activity of the sympatho-adrenal system and monoamine oxidase (MAO) in women diagnosed with microvascular angina. A total of 28 women were examined and divided into three groups. Group I, the control group, consisted of 10 healthy women; Group II included 8 women diagnosed with stable exertional angina of functional class II; and Group III included 10 women with microvascular angina.
According to the obtained results, patients with microvascular angina demonstrated increased activity of the sympatho-adrenal system. It was also established that the MAO enzyme, which is responsible for the deamination of catecholamines, showed a significant decrease in its activity. This condition is believed to result from increased lipid peroxidation processes, which lead to enhanced and prolonged toxic effects of catecholamines on the myocardium.

Keywords: microvascular angina, sympatho-adrenal system, catecholamines, monoamine oxidase, lipids

INTRODUCTION: Microvascular angina (MVA), one of the clinical forms of ischemic heart disease (IHD), remains a significant cause of mortality despite advances in understanding its pathophysiology and pathogenesis, as well as the development of modern diagnostic and treatment methods. The rates of death and disability caused by IHD continue to increase.

In women, the main factors contributing to these diseases include constant psycho-emotional stress, atherosclerosis, hyperlipidemia, physical inactivity, diabetes mellitus, arterial hypertension, obesity, and menopausal conditions [1,3,9,11,14].

Despite differing opinions, the results of clinical studies and autopsies presented in the literature suggest that functional ovarian activity provides a protective effect against the development of IHD. However, several researchers question the hypothesis of ovarian–estrogen protection and instead propose another theory — that men around the age of 50 lose a factor that had previously delayed the progression of IHD.

Modern population studies indicate that in recent years, mortality from cardiovascular diseases among women has not decreased but continues to rise. The main causes of death in women are IHD, arterial hypertension, and cerebrovascular disorders. According to the Framingham study, after menopause, the risk of developing cardiovascular pathology in women is 2–3 times higher compared to women of the same age who have not yet reached menopause.

The increased prevalence of these diseases is associated with estrogen deficiency during the climacteric period. Estrogens affect peripheral blood vessels in the same way as they influence coronary arteries. Their effect is mainly due to their role as endogenous factors promoting vascular relaxation, primarily through increased nitric oxide levels. Estrogens improve endothelial function, reduce smooth muscle cell proliferation in response to vascular injury, and maintain vascular integrity.

During menopause, estrogen deficiency leads to the development of endothelial dysfunction, which contributes to the emergence of microvascular forms of IHD and arterial hypertension. Estrogens accelerate the metabolism of low-density lipoproteins (LDL) and increase the production of bile acids. They slow down the deposition of LDL, reduce total cholesterol levels, and decrease the amount of α-lipoproteins and β-lipoproteins. Estrogens are also capable of reducing the inflammatory processes associated with the development of atherosclerosis.

The vasodilatory and antiatherogenic effects of female sex hormones are related to their antagonistic properties. Experimental data indicate that estrogens inhibit platelet aggregation, stimulate the synthesis of elastic and collagen fibers, and also influence the level of hemosiderin. Additionally, they increase insulin secretion and sensitivity to insulin.

A deficiency of estrogens deepens vascular spasticity and platelet aggregation, increases plasma renin levels, and elevates both the amount of angiotensin II and the sensitivity of its receptors. During this period, age-related changes occur in the cardiovascular system of women, mainly the onset of atherosclerosis and elevated arterial blood pressure, which in turn reduces the adaptive capacity of the cardiovascular system.

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In the pathogenesis of microvascular angina (MVA), a major role is also played by changes in the functional state of the sympatho-adrenal system (SAS), which are associated with increased sensitivity of receptor apparatuses. A marked activation of lipid peroxidation (LPO) processes has also been observed. In ischemic heart disease, an increase in the levels of triglycerides (TG), free fatty acids, and cholesterol (Ch) in the blood is commonly noted [3,5,11,12,13]. At present, in modern medicine, data on the interrelationship between the functional state of the SAS, the activity of monoamine oxidase (MAO), and their alterations in MVA are still insufficient. Moreover, in women diagnosed with MVA during the menopausal period, these aspects have been studied only minimally.

PURPOSE OF THE STUDY: To investigate the metabolism of catecholamines and the functional state of the antioxidant system in women diagnosed with microvascular angina.

MATERIALS AND METHODS: To assess the functional state of the sympatho-adrenal system (SAS) in patients with microvascular angina (MVA), the concentrations of adrenaline (A) and noradrenaline (NA) in blood serum, as well as the urinary excretion of catecholamines (CA), including A, NA, dopamine (DA), and DOPA, were measured.

A total of 28 women aged 49–69 years participated in the study. The diagnosis of MVA was confirmed based on comprehensive clinical and instrumental examinations. All participants underwent clinical and laboratory tests, electrocardiography (ECG), and radiological assessments.

The women were divided into three groups:

- Group I – Control group (10 healthy women)
- Group II – Women with stable exertional angina of functional class II (8 women)
- Group III – Women with microvascular angina (10 women)

The daily urinary excretion of catecholamines was measured using the trioxinol fluorometric method according to the modification by E.Sh. Matlin, Z.M. Kiseleva, and I.E. Sofieva. The total content of conjugated catecholamines in urine was determined using the method of T.I. Lukicheva, V.V. Menshikov, and T.D. Bolshakov. The activity of monoamine oxidase (MAO) in the blood was evaluated according to the method of A.I. Balakleevsky. The lipid spectrum was studied using a modern biochemical express analyzer (Reflotron-Roche).

RESULTS:

Table 1. Daily urinary excretion of catecholamines and monoamine oxidase (MAO) activity in the examined women

CA	Fractions	Group I (Control)	Group II	Group III
A, µg /day	Free	4,5±0,1	5,0±0,1	5,8±0,1
	Conjugated	3,8±0,1	4,8±0,1	5,0±0,12
	Total	8,3±0,2	9,8±0,1	10,8±0,2
NA, µg /day	Free	9,9±0,1	10,8±0,07	11,4±0,08
	Conjugated	8,7±0,1	9,6±0,08	10,5±0,09
	Total	18,6±0,2	20,4±0,1	21,9±0,08
DA, µg /сут	Free	140,4±4,2	80,9±1,5	157,6±2,5
	Conjugated	152,8±5,3	98,4±2,4	168,7±3,4
	Total	293,2±5,5	179,3±1,8	326,3±2,8
DOPA, µg /day		46,4±0,6	35,4±0,8*	48,6 ±0,6
A/NA ratio, units		0,44	0,49**	0,43**
NA/DA ratio, units		0,063	0,067*	0,09
DA/DOPA ratio, units		6,32	6,58	4,19
MAO, units/ex		0,06±0,002	0,05±0,003	0,04±0,004

Note: Free – unconjugated form; Conj. – conjugated form; Total – combined (sum) value. **Explanation:** * – statistically significant compared with Group I ($p = 0.05$); ** – not statistically significant ($p > 0.05$).

Table 2. Serum lipid spectrum in the examined women

Indicators	Control group	Group II	Group III

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Total cholesterol (mmol/L)	3.4 ± 0.3	7.7 ± 0.1 ($p_1 < 0.001$)	8.44 ± 0.14 ($p_1 < 0.001$, $p_2 < 0.001$)
LDL cholesterol (mmol/L)	2.42 ± 0.3	5.9 ± 0.06 ($p_1 < 0.001$)	6.72 ± 0.11 ($p_1 < 0.001$, $p_2 < 0.001$)
HDL cholesterol (mmol/L)	1.3 ± 0.1	1.05 ± 0.03 ($p_1 < 0.001$)	1.0 ± 0.03 ($p_1 < 0.001$, $p_2 < 0.05$)
VLDL cholesterol (mmol/L)	0.28 ± 0.02	0.72 ± 0.02 ($p_1 < 0.001$)	0.82 ± 0.03 ($p_1 < 0.001$, $p_2 < 0.05$)
Triglycerides (mmol/L)	1.1 ± 0.1	1.6 ± 0.04 ($p_1 < 0.001$)	1.8 ± 0.06 ($p_1 < 0.001$, $p_2 < 0.001$)
Atherogenic index (units)	2.1 ± 0.1	6.6 ± 0.22 ($p_1 < 0.001$)	7.85 ± 0.18 ($p_1 < 0.001$, $p_2 < 0.001$)

Note:
p_1 – statistically significant difference between Group I and Group II;
p_2 – statistically significant difference between Group II and Group III. The lipid spectrum parameters of the blood differed significantly from those of the control group ($p < 0.001$).

In our study, when evaluating lipid metabolism changes in women diagnosed with microvascular angina (MVA), it was observed that in healthy women (control group), the level of total cholesterol (TC) was up to 3.4 ± 0.3 mmol/L. In women of Group II, the levels of TC increased by 38.1%, triglycerides (TG) by 28.4%, low-density lipoproteins (LDL-C) by 41.3%, and the atherogenic coefficient by 38.5%, while high-density lipoproteins (HDL-C) decreased by 17.3% compared with the control group.

In Group III, women with MVA showed more pronounced changes: TC increased by 59%, TG by 37.3%, LDL-C by 68.4%, HDL-C decreased by 21.4%, and the atherogenic index increased by 44% compared with the control group. When comparing the data of Groups II and III, it was found that patients with MVA had slightly higher absolute values, with key biochemical markers maximally elevated. In this group, TC reached 8.44 ± 0.14 mmol/L, and LDL-C reached 6.72 ± 0.11 mmol/L. The increase of TC was 19.5% (1.17-fold), and LDL-C was 20.2% (1.16-fold) higher in Group III compared with Group II.

Thus, the obtained results demonstrate that in patients with MVA, lipid metabolism is significantly disturbed, which is reflected by increased levels of TC, LDL-C, and atherogenic index, along with a decrease in HDL-C. The degree of hypercholesterolemia and hyperlipoproteinemia tends to rise in parallel with the severity of ischemic heart disease (IHD) and with advancing age.

When assessing the daily excretion of catecholamines (CA), the following changes were revealed. In the control group, adrenaline (A) and noradrenaline (NA) excretion levels were within normal limits. According to the findings, women in Group II showed an increase in A: free fraction by 30.6%, conjugated by 58.5%, and total by 42.4%; NA: free by 5.8%, conjugated by 43.1%, and total by 23.7%. Dopamine (DA) and DOPA levels showed decreases: DA free by 42.5%, conjugated by 5.5%, and total by 25.6%; DOPA by up to 16.4% lower than in the control group ($p > 0.05$).

In women of Group III, the daily excretion of catecholamines and DOPA was markedly elevated on the first day of observation. Total adrenaline (A total) reached 10.8 ± 0.2 μg/day, which was 29.8%, 33.4%, and 31.7% higher than the control values ($p < 0.001$). The daily urinary excretion of NA also showed a statistically significant increase: total NA (NA total) reached 21.9 ± 1.1 μg/day, which was 16.5%, 22.2%, and 19.7% higher than in the control group ($p < 0.001$). In all fractions, the daily excretion of dopamine (DA) was statistically significantly higher compared with the control group. The total DA excretion (DA total) reached 326.3 ± 6.0 μg/day, which was 19.5% higher than that of the control group ($p < 0.001$). The daily excretion of DOPA was 49.6 ± 0.8 μg/day, exceeding the control values by 9.2% ($p < 0.05$). To study the activity of the sympatho-adrenal system (SAS) in patients with ischemic heart disease (IHD) more comprehensively, it was of particular interest not only to assess the quantitative indicators of catecholamines (CA) and DOPA in the urine but also to evaluate the adequacy of the ratio of these metabolites to each other. For this purpose, the comparative coefficients proposed by T.D. Bolshakova were used, which reflect the relationships between the metabolic products in the CA metabolic chain:

- DA/DOPA – characterizes the biosynthesis of DA from DOPA;
- NA/DA – reflects the biosynthesis of NA from DA;
- A/NA – indicates the biosynthesis of A from NA;

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- (Ae + Ab)/Ac, (NAe + NAb)/NAc, (DAe + DAb)/DAc – characterize the processes of conjugation of A, NA, or DA with sulfate residues.

According to the obtained results, slight variations in the comparative coefficients were observed. On the first day of the study, the DA/DOPA ratio indicated normal DA biosynthesis. We observed an increase in the NA/DA ratio, suggesting a potential activation of NA biosynthesis. The A/NA ratio increased by 14%, indicating enhanced synthesis of adrenaline.

When studying the activity of monoamine oxidase (MAO) in the blood serum, it was found that in women with microvascular angina (MVA), MAO activity was reduced compared to healthy women: by 30% in Group I, by 21.6% in Group II, and by 46% in Group III ($p < 0.05$).

ANALYSIS: In microvascular angina (MVA), changes in the functional activity of the sympatho-adrenal system (SAS) depend on multiple physiological and pathological processes. The central nervous system plays a leading role among the body's regulatory systems. An increase in the functional activity of the SAS, particularly in the early stages of the disease, acts as a compensatory mechanism aimed at enhancing energy supply. However, in later stages, its metabolic effects become more pronounced. The higher the activity of this system, the greater the risk of developing ischemic heart disease (IHD). Hormonal changes in the female body can lead to serious cardiovascular consequences. These include weight gain, elevated plasma lipid levels, disturbances in sympathetic activity, and vascular dysfunction — all of which act as primary complex mechanisms contributing to cardiovascular pathology [2,4,8,10]. Disturbances in monoamine oxidase (MAO) activity lead to the accumulation of lipid peroxidation (LPO) products. According to our study results, MAO activity was reduced. During the process of lipid peroxidation, MAO, the main enzyme responsible for the transformation of biogenic amines, becomes inactivated, which in turn decreases its activity toward monoamines. In women with ischemic heart disease, the increased functional activity of the SAS enhances the negative effects of catecholamines through excessive stimulation of β-adrenergic receptors. Based on our results, in menopausal women with microvascular angina, both intensification of MAO inhibition and increased activity of the SAS were observed.

The findings of our study may partially explain the early development of MVA in women and can contribute to the early diagnosis of ischemic heart disease and the prevention of its adverse outcomes.

CONCLUSION: Thus, in women diagnosed with microvascular angina, comprehensive clinical evaluation of catecholamine metabolism revealed an increase in the daily urinary excretion of catecholamines. This finding indicates a dysfunction in the functional state of the sympatho-adrenal system.

It was also determined that in women with microvascular angina, particularly during the climacteric and menopausal periods, monoamine oxidase (MAO) activity was significantly reduced. A marked decrease in MAO activity reflects alterations in the catalytic properties of the enzyme in this group of patients, which consequently leads to enhanced and prolonged toxic effects of catecholamines on the myocardium.

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