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Original Research

Study of indicators of intracardial hemodynamics and structural state of the myocardium in monotherapy of patients with arterial hypertension with moxonidin

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ABSTRACT:

A total of 61 hypertensive patients with II-III degrees of increased blood pressure (WHO, 1999) aged 33 to 74 years (mean age 54.5 + 1.3) with a disease duration of 3 to 15 years were examined. Arterial hypertension of II degree occurred in 32 (52.5%), III degree - in 29 (47.5%). Among the surveyed men - 29, women - 32. All clinical and laboratory-instrumental studies were carried out on the basis of the SamMI clinic in the departments of cardiology and therapy. We carried out the selection of patients, clinical and instrumental laboratory examination, dynamic observation. The diagnosis of hypertension was made on the basis of anamnesis, clinical data and examination results according to the generally accepted program of two-stage examination of patients.

Thus, we can conclude that, against the background of 12-week therapy with moxonidine in patients with arterial hypertension, there is a moderate significant regression of left ventricular hypertrophy, associated, first of all, with a decrease in the thickness of the posterior wall of the left ventricle and the thickness of the interventricular septum and positive changes in the parameters of intracardiac hemodynamics.

Key words: hemodynamics, cardiology, myocardium, blood pressure, arterial hypertension

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INTRODUCTION

Arterial hypertension occupies one of the leading places in the structure of cardiovascular diseases. The prevalence of arterial hypertension is 15-20% [1]. The presence of arterial hypertension is associated with an increased risk of coronary heart disease, cerebral stroke, heart and renal failure, and general mortality.

As is known, in patients with arterial hypertension, the overall mortality rate is 2-5 times higher, and mortality from cardiovascular diseases is 2-3 times higher [2].

In more than 90% of cases, arterial hypertension is primary, i.e. we are talking about hypertension [3]. With the development of left ventricular hypertrophy in patients with arterial hypertension, the life prognosis is significantly worse, since it is accompanied by a fivefold increase in the risk of sudden death, and in general, the survival rates deteriorate. In the MRFIT study (1996), it is with left ventricular hypertrophy that an increase in mortality from complications of arterial hypertension is associated. The negative effect of left ventricular hypertrophy on survival is associated with a decrease in coronary reserve, impaired endothelial function, the occurrence of arrhythmias, the development of diastolic and later systolic dysfunction of the left ventricle [4,5,6].

According to the views [7,8], in the vasomotor centers of the medulla oblongata, two types of receptors on the membranes of neurons mediate the regulation of

the tone of the sympathetic nervous system: α2-adrenergic and I1-imidazoline.

Initially, it was believed that α2-adrenergic receptors and I1-imidazoline receptors are located on the presynaptic membrane of the same neurons and regulate the release of norepinephrine through a negative feedback mechanism. According to modern concepts, imidazoline receptors are an independent type of receptors, represented by two subtypes: I1 and I2 [9].

It is assumed that I2 receptors are localized in mitochondria, they are somehow interconnected with monoamine oxidase and are involved in the pathogenesis of depression, in the development of Alzheimer's disease and Parkinson's disease, and play a role in hyperalgesia in acute inflammation.

I1 receptors, considered as functionally active membrane receptors, have been studied more [10].

Imidazoline I1 receptors are localized in the ventrolateral part of the rostral part of the brainstem (rostral ventrolateral medulla), which is the center of blood pressure regulation, as well as the kidneys, adrenal glands, pancreatic islets of Langerhans, fat cells (adipocytes) and the carotid glomerulus [9]

Agonism in relation to I1-imidazoline receptors of neurons located in the ventrolateral nuclei of the medulla oblongata is accompanied by the development of an antihypertensive effect [10]. In addition to lowering blood pressure, stimulation of I1-imidazoline receptors is accompanied by a decrease in heart rate.

In recent years, the mechanisms of development of left ventricular diastolic dysfunction and its role in the onset of chronic heart failure have become the subject of research of clinicians [11, 12, 13, 14]. Until now, isolated left ventricular diastolic dysfunction was traditionally considered the mechanism of chronic heart failure in patients with preserved left ventricular systolic function. However, there are isolated studies indicating that in patients with symptoms of chronic heart failure, but without signs of impaired global systolic function of the left ventricle according to two-dimensional echocardiography, tissue Doppler echocardiography reveals impaired longitudinal systolic function of the left ventricle [12,13].

MATERIALS AND METHODS

A total of 61 hypertensive patients with II-III degrees of increased blood pressure (WHO, 1999) aged 33 to 74 years (mean age 54.5 + 1.3) with a disease duration of 3 to 15 years were examined.

Arterial hypertension of II degree occurred in 32 (52.5%), III degree - in 29 (47.5%). Among the surveyed men - 29, women - 32.

All clinical and laboratory-instrumental studies were carried out on the basis of the SamMI clinic in the departments of cardiology and therapy. We carried out the selection of patients, clinical and instrumental laboratory examination, dynamic observation. The diagnosis of hypertension was made on the basis of anamnesis, clinical data and examination results according to the generally accepted program of two-stage examination of patients.

The exclusion criteria were: patients with EH with an ejection fraction of less than 50%, patients with symptomatic arterial hypertension, unstable angina pectoris and angina pectoris III-IV functional classes, signs of stage IIB-III heart failure, valvular heart disease, pulmonary pathology, severe cerebrovascular accident (hypertensive encephalopathy stage II, transient disorders of cerebral circulation), as well as other organic lesions.

The control group consisted of 15 apparently healthy individuals (7 men and 8 women), average age 49.5 + 1.7 years.

For 7-10 days (control period), all antihypertensive drugs were discontinued. Before the start of program research, along with general clinical research, all patients underwent laboratory and instrumental examination.

According to the study protocol, all patients were prescribed moxonidine. With the help of echocardiographic study, the functional state of the myocardium, indicators of contractile function, the nature of myocardial remodeling in patients with arterial hypertension were studied.

RESULTS AND DISCUSSION

It was shown that in patients with EH at the initial stage of observation, the volume indices of the left ventricle did not significantly differ from the control values, however, there was a tendency (in the group as a whole) to increase the end-diastolic and end-systolic volumes. The left ventricular ejection fraction remained within the control values. At the same time, a significant increase in the thickness of the myocardial walls was noted in comparison with the healthy group. Thus, the thickness of the interventricular septum was increased to 1.34 + 0.03 cm versus 0.98 + 0.02 cm ($p < 0.001$), the thickness of the posterior wall of the left ventricle was increased to 1.12 + 0.02 cm versus 0.88 +0.01 cm ($p < 0.001$). Along with this, when determining the indexed mass of the left ventricular myocardium, the values of the mass index of the left ventricular myocardium were obtained, amounting to 137.7 + 6.1 g / m2 versus 101.1 + 5.0 in the control ($p < 0.001$).

Patients with arterial hypertension with different types of blood circulation were characterized by the absence of differences in the myocardial mass of the left ventricle: the mass index of the left ventricular myocardium in the normokinetic type of blood circulation was 130.6 + 5.9 g / m2, in the hyper- and hypokinetic type -148 + 7.19 g / m2 and 132.8 + 6.29 g / m2 ($p < 0.05$ compared to control for all cases).

Taking into account the value of the left ventricular myocardial mass index, signs of left ventricular hypertrophy were detected in 34 of 61 patients (55.7%). We determined the frequency of the following types of left ventricular hypertrophy:

concentric type - in 21 (34.4%), eccentric type - without dilatation - in 10 (16.4%), eccentric type with dilatation - in 3 (4.9%) In the absence of left ventricular hypertrophy (with a left ventricular myocardial mass index <134 g / m2), the following types of left ventricular myocardial geometry were identified: normal geometry - in 11 (18.0%), concentric remodeling - in 5 (8.2%)), isolated hypertrophy of the interventricular septum - in 7 (11.5%), isolated hypertrophy of the posterior wall of the left ventricle - in 4 (6.6%).

During 12-week therapy with moxonidine, a characteristic change in the types of geometry of the left ventricle was noted with a tendency to a decrease in the severity of concentric hypertrophy and the frequency of eccentric type of geometry of the left ventricle with dilatation. Thus, by the end of the course of observation, 3 patients with concentric left ventricular hypertrophy (at the initial stage) showed a transformation into a concentric type of remodeling; in 2 patients with eccentric left ventricular hypertrophy with dilatation by the end of treatment, its transformation into eccentric left ventricular hypertrophy without dilatation took place; 1 patient with a concentric type of remodeling and 2 patients with isolated hypertrophy of the interventricular septum revealed a transformation into a normal type of left ventricular geometry.

Thus, by the end of therapy, left ventricular hypertrophy was observed in 31 (50.8%) versus 34 (55.7%) at the initial stage. At the same time, concentric left ventricular hypertrophy was noted in 18 (29.5%), eccentric type of left ventricular hypertrophy with dilatation in only 1 (1.67%), eccentric type of left ventricular hypertrophy without dilatation - in 12 (19.7%) ... The distribution of the geometry types of the left ventricle also changed: normal geometry was found in 14 (22.9%), concentric remodeling - in 7 (11.5%), isolated hypertrophy of the interventricular septum - in 5 (8.2%), the posterior wall of the left ventricle - 4 (6.6%).

The revealed dynamics in relation to the types of geometry of the left ventricle was accompanied by certain structural changes in the left ventricular myocardium.

In the group as a whole, a mild tendency towards a decrease in end-diastolic volume (by 3.4□3.1%) and end-systolic volume (by 3.7□2.0%) was revealed, the stroke volume remained practically unchanged, the same as an ejection fraction: 57.6 - 1.7% versus 56.5 - 1.4% at the initial stage. There was a significant decrease in the thickness of the interventricular septum, the thickness of the posterior wall of the left ventricle, the mass index of the left ventricular myocardium, however, the average values of these parameters remained significantly increased compared with the control group.

Subsequently, taking into account the volumetric parameters of the left ventricle, 2 groups were identified: group 1 (n = 21) included patients with an initial level of end-diastolic volume> 130 ml, the remaining patients were group 2 (n = 40).

The analysis of the data obtained showed that in patients of the 1st group with the initial values of end-diastolic and end-systolic volumes: 157.1±8.1 cm3 and 71.0±5.9 cm3, respectively, in contrast to patients of the 2nd groups with initial values of end-diastolic and end-systolic volumes: 120.1±4.9 cm3 and 51.9±3.7 cm3, respectively, there was a more pronounced dynamics of the analyzed parameters. So, end-diastolic volume in group 1 was - 6.5±3.9% versus + 0.3±0.9%. A similar trend was found for CSR: in the 1st group, CSR was - 6.8 3.9% versus - 1.9±1.2%.

At the same time, the increase in the ejection fraction in the 1st group was also slightly higher than in the 2nd group: +3.7±1.9% versus 1.4±0.9%.

We also considered it necessary to study the dynamics of echocardiographic parameters of left ventricular hypertrophy during therapy with moxonidine. For this purpose, a group of patients with left ventricular hypertrophy was identified. At the same time, the initial thickness of the interventricular septum and the posterior wall of the left ventricle were 1.47±0.03 cm and 1.14 ±0.02 cm, respectively, and the left ventricular mass index was -148.4 ±6.7 g / m2. After 12 weeks of therapy with moxonidine, a significant change in the mean values of the studied parameters was noted: a decrease in the thickness of the posterior wall of the left ventricle to 1.08 + 0.03 cm; the thickness of the interventricular septum to 1.4 + 0.03 cm, the mass index of the left ventricular myocardium to 140.5 + 5.9 g / m2 ($p < 0.05$ in all cases), while the mass index of the left ventricular myocardium decreased by 5.5 %. Taking into account the measurement error when performing echocardiography, the generally accepted criterion for regression of left ventricular hypertrophy is a decrease in the left ventricular myocardial mass index by 10%. In this regard, our results were regarded as a positive trend towards regression of the left ventricular myocardium mass during 12 weeks of moxonidine therapy.

CONCLUSION

Thus, we can conclude that, against the background of 12-week therapy with moxonidine in patients with arterial hypertension, there is a moderate significant regression of left ventricular hypertrophy, associated, first of all, with a decrease in the thickness of the posterior wall of the left ventricle and the thickness of the interventricular septum and positive changes in the parameters of intracardiac hemodynamics.

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CONSENT

Written informed consent was obtained from all participants of the research for publication of this paper and any accompanying information related to this study.

CONFLICT OF INTEREST

The authors declare that they have no competing interests.

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