



Immuno-Inflammatory and Hormonal Status with Arterial Hypertension

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Abstract: In women suffering from arterial hypertension of the 1st and 2nd degree, the state of the immunoinflammatory and hormonal status was studied. The study involved 90 patients aged 45-74 years. 30 patients suffered from grade 1 hypertension, 30 patients suffered from grade 2 hypertension, and 30 patients made up the control group. Examined in all patients PCT, ng/ml, IGF-I, ng/ml, VEGF, pg/ml, TGF- β 1, pg/ml.

Keywords: hypertension, hormonal status, immunoinflammatory status, PC, IGF-I, VEGF, TGF- β 1.

According to the Congress of the European Society of Cardiology, CVD mortality in women (55.0%) is significantly higher than in men (43.0%). The awareness of patients about the presence of AH is 77.9% [3].

One of the promising areas for improving the quality of diagnosis and stratification of patients according to the severity of the condition in scientific and practical studies is the determination of the concentration of procalcitonin (PCT), a marker of a systemic inflammatory response and bacterial infection [1].

Purpose of the study: Estimation and immunoinflammatory and hormonal status in women with arterial hypertension

Materials and research methods.

90 patients were under observation, of which 30 women (group 1) with stage 1 hypertension, 30 women (group 2) with stage 2 hypertension. The control group consisted of 30 healthy women (control group) aged 45-74 years. Examined in all patients PCT, ng /ml, IGF - I , ng /ml, VEGF , pg /ml, TGF - β 1 , pg/ml.

Results and its discussion.

The study of biochemical mediators of inflammation in women with AH showed an increase in the level of procalcitonin (PCT) regardless of the degree of AH.

At the same time, the threshold level, indicating the development of inflammation in women with stage 1 hypertension, is a PCT concentration of 0.05 ng / ml. In patients with concomitant diseases with 1-degree AH, the concentration of PCT was significantly higher than with AH -2 degree, which confirms the influence of concomitant diseases on the development, course, and outcome of AH.

It was important to study the effect of growth hormones on the course of hypertension depending on sex.

It is known that insulin-like growth factor (IGF-I), as a polypeptide hormone, consists of small protein molecules. They are also necessary for the normal growth and development of bones and tissues of the body. IGF-I is synthesized in the liver and skeletal muscles as a result of their stimulation by growth hormone.

IGF-I, like a tropic hormone, activates the reparative mechanisms of vascular wall repair, thereby protecting and preventing the processes of damage to the vascular wall [8].

An increase in the concentration of IGF-I in CAD can be considered as a mechanism for activating the processes of damage and protection of the vascular wall and as an indicator of “instability of atherosclerotic plaques”, which means an increased risk of outcome in MI [1,4].

Taking into account the above data, a study was conducted to determine the level of IGF-I in the blood of the examined patients, depending on the degree of hypertension.

As a result, a significant increase in its concentration by 1.28 times was found in AH of the 1st degree - up to 123 ± 2.49 ng /ml and by 1.43 times in AH 2-degree up to 136.6 ± 1.25 ng /ml against control values - 95.5 ± 1.8 ng /ml ($P < 0.001$).

The results obtained confirm the process of damage to the vascular wall and activation of protection. At the same time, an increase in the concentration of IGF -I in the blood was revealed, depending on the severity of the course, the more severe the course of hypertension, the more elevated IGF-I in the blood.

Therefore, in women with hypertension, in response to damage to the vascular wall as a result of dyslipidemia, there is an increase in IGF-I in the blood. When predicting the outcome of hypertension in women, it is necessary to take into account the metabolic status and concomitant diseases, including CVD.

Thus, the mechanism of CVD development is closely related to vascular endothelial dysfunction. The degree and severity of hypertension directly depends on the degree of damage to the vascular wall and myocardium. The activity of growth factors and inflammatory markers has an important prognostic value for assessing the progression of CVD and the development of angiopathy .

According to the literature, it is known that the endothelium of the vascular intima performs barrier, secretory, hemostatic, and vasotonic functions, plays an important role in the processes of inflammation and remodeling of the vascular wall [28].

There is evidence that body aging is accompanied by the development of endothelial dysfunction and is accompanied by an increase in the concentration of endothelin [7].

Other authors have shown that in young people (from 25 to 41 years old) the levels of endothelin-1 correlate with risk factors for the development of cardiovascular pathology [8].

Vascular endothelial growth factor (VEGF) in AH was determined . At the same time, an increase in the level of VEGF in AH of the 1st degree to 296 ± 16.53 pg /ml was found, which is 3.2 times more than the control indicators - 92.4 ± 1.5 pg /ml ($P < 0.001$, Table 1) .

Table No. 1. Growth and damage factors in hypertension

Blood indicators	1-control group n=30	Women n= 60	
		1-gr n=30	2-gr n=30
PCT, ng /ml	0.03 ± 0.02	0.2 ± 0.01 *	0.2 ± 0.01 *
IGF-I , ng /ml	95.5 ± 1.8	123.0 ± 2.49 *	136.6 ± 1.25 ***
VEGF , pg /ml	92.4 ± 1.5	296 ± 16.53 ***	426.2 ± 13.34 ***
TGF- β 1 , pg/ml	10.0 ± 1.5	22.0 ± 0.94 *	15.5 ± 0.58 *

Note: * Values are significant in relation to the control group ($P < 0.05 - 0.001$)

In AH of the 2nd degree of severity, its concentration was increased by 4.6 times (up to 426.2 ± 13.34 pg /ml) against the control - 92.4 ± 1.5 pg /ml ($P < 0.001$).

As a result, data were obtained indicating the process of inflammation of the vascular wall in AH and a direct dependence of the activity of inflammation on the severity of AH.

Consequently, the duration and severity of hypertension in women aged 50 years and older determine the involvement of the myocardium and brain in the process, with the subsequent development of cardiogenic and cerebral complications. At the same time , the threshold level,

indicating the development of cardiogenic and cerebral complications in hypertension in women, is the concentration of VEGF -182.5 pg / ml.

One of the most important pathogenetic factors that lead to the activation of fibrosis and myocardial hypertrophy processes is transforming growth factor-1 (TGF- β 1). Under experimental conditions in the culture of cardiomyocytes, its role in the development of their hypertrophy was shown. The activation of TGF- β 1 is associated with the phenotypic heterogeneity of myofibroblasts, and hence the possibility of their formation of connective tissue, since these cells are capable of synthesizing glycoproteins and all types of collagens, as well as matrix-modifying proteins. The action of this growth factor is associated with the development of interstitial fibrosis, as well as a decrease in the elastic properties of the myocardium and blood vessels [8].

Fibroblast growth factor (unlike VEGF) is not only specific for endothelial cells; its receptors have been found on many other cells, including fibroblasts and vascular smooth muscle cells. FGF (as well as VEGF) stimulates many endotheliocyte functions, such as proliferation, migration, extracellular proteolytic activity, and tubule formation [1].

The most important role of TGF- β 1 in the development of pathological left ventricular remodeling in HD is quite clearly substantiated in the literature by data on powerful profibrogenic and hypertrophic effects of this factor. Moreover, TGF- β 1 is now considered as the main factor initiating and realizing the formation of myocardial fibrosis. It has been proposed that excessive production of transforming growth factor- β 1 (TGF- β 1) is involved in the long-term consequences of hypertension, including left ventricular hypertrophy (LVH), 1,2-vascular remodeling and progressive kidney disease [3].

In our studies, a statistically significant increase in the level of TGF- β 1 to 22 ± 0.94 pg /ml in patients of the 1st group and up to 15.5 ± 0.58 pg /ml in patients of the 2nd group was established against the control values of 10.0 ± 1.5 pg /ml ($p < 0.05$).

The results of the analyzes showed the threat of developing CVD and fibrosis, LV hypertrophy in AH. At the same time, the concentration of TGF- β 1 does not depend on the severity of AH, since the level of TGF- β 1 is more elevated in 1-degree AH than in 2-degree AH.

Therefore, the course and outcome of hypertension in women depends on comorbidities and metabolic status. Regardless of the degree of hypertension, in women, an increase in TGF- β 1 in the blood indicates the threat of fibrosis and myocardial hypertrophy.

Conclusion

Thus, the mechanism of development of hypertension in women is closely related to vascular endothelial dysfunction. The degree and severity of hypertension directly depends on the degree of damage to the vascular wall and the state of metabolism in the myocardium.

Postmenopausal women have a high risk of developing and progressing CVD, namely, fibrosclerosis and myocardial hypertrophy against the background of hypertension, regardless of severity. For early detection of the risk of developing CVD in patients with hypertension, regular examination and study of the activity of growth factors and pro-inflammatory markers for the prevention of angiopathy and coronary artery disease is necessary.

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