CELLULAR FACTORS OF ENDOTHELIAL DEVELOPMENT DYSFUNCTIONS AT NSA INTRODUCTION

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Non-specific aortoarteritis (NSA) is known to be a common systemic vasculitis affecting the aorta and large major vessels. NSA more often affects middle-aged people from 30 to 40 years old, and in these patients, NSA acts as a risk factor for the development (RDF) of atherosclerosis, coronary heart disease, heart failure in 74% of patients with NSA, arterial hypertension (AH), the leading clinical syndrome. The etiology of AH can be hemodynamic disturbances, cerebral ischemia, as well as other nosologies. AH in patients is a powerful RDF of formation and pathology from both the heart and blood vessels [1, 3, 5].

An important role in the dynamics in the status of the vascular wall (VW) is assigned to the inner layer of blood vessels - it is anatomically called endothelium and it is important to emphasize: it is the main "target organ" in NSA. When exposed to destructive agents, the compensatory function of the vascular endothelium is disturbed, which contributes to the destruction of physiological, biochemical and immunological mechanisms in VW [1, 2, 4, 6].

The main objective of this work is to study the relationship between the T- and B-cell component of the immune system (TBCIS) and the physiological status (PS) of the vascular endothelial wall (VEW) of patients with NSA.

Materials and research methods

81 patients with moderate, II-stage NSA activity aged 21 to 43 years were examined. Of these, 55 people had type I arterial damage, and 26 - type III. All patients had AH (46 people had stage II, 35 people had stage III). The control group included 30 healthy donors to compare data. Lymphocytes were phenotyped using an indirect immunoperoxidase test using monoclonal antibodies (Sorbent-Service LLP, Moscow) to surface structures CD4, CD8, CD20, CD25, CD95, HLA-DR. Characterization of VEW and its functions was performed using migrating endothelial cells (MEC), quantitative values of ETN-1 (endothelin-1) in the blood of patients, and assessment of endothelial-dependent vascular relaxation (EDVR) of the arteries of the shoulder region. MEC assessment conducted according to J. Hladovec (1978). ETH-1 values were detected by IEA (Biomedica). Standard statistical processing and correlation analysis of the obtained values was carried out using the program “Statistica 6.0”.

Results and discussion

TBCIS values in patients with NSA are given in table 1. From the data table 1 implies the following: in NSA, there are disproportions in the subpopulation of lymphocytes, in contrast to the control. So, for example, in the examined NSA with stage II AH, an increase in the relative number of T-helpers and suppressor lymphocytes was revealed - these are CD4 + and CD8 + cells. In patients with NSA with III tbsp. AH showed an increase in levels of CD4 + and CD8 + - lymphocytes, in contrast to those in patients with stage II NSA and AH. In the study of the B (CD20 +) component, an increase in the

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relative level of CD20 + cells in the blood of patients with NSA was noted, which tended to increase along with the severity of AH.

Table 1. TBCIS in the peripheral blood of patients with NSA (M±m).

Groups	Surface markers (%)					
	CD4+	CD8+	CD20+	CD25+	CD95+	HLADR+
Control	38,1 ± 3,1	22,1 ± 2,3	11,6 ± 2,2	13,8 ± 1,4	21,4 ± 1,8	18,4 ± 4,8
NSA + AH II stage	60,1 ± 2,5 *2	28,9 ± 2,1 *2	20,9 ± 1,3	29,2 ± 1,2	39,1 ± 2,4	20,9 ± 2,5
NSA + AH III ge	67,7 ± 3,3 *1-3	16,6 ± 2,5 *1-3	24,3 ± 1,4	34,3 ± 1,4	44,8 ± 2,9 *1-	36,1 ± 1,9
Note: *–(p<0,05) – statistically significant differences from the control and other groups.						

In patients with NSA, increased expression of an early marker of lymphoid cell stimulation, the α-chain of IL-2 receptor (CD25 +) and the expression of final activation markers (HLA-DR +), which was comparable to stage AH, were determined. In the same category of patients, an increase in apoptosis factor CD95 + increased, which increased along with the stage of AH. The analysis performed revealed a deepening of the destruction of TBCIS in the subjects diagnosed with NSA, which correlated with the degree of AH III.

The most pronounced shifts in the number of CD4 +, CD8 + –cells, an increase in the relative number of B-lymphocytes with the CD20 + phenotype, as well as inversion of the expression of early CD25 + and late HLA-DR + predictors of stimulation and induction of apoptotic factor CD95 + were a hallmark in NSA patients with maximum AH stage, with III degree (table 1).

Damaged due to immupatogenesis and inflammation, NSA VEW exerts its damaging effect in the form of an increase in ETN-1 production and a decrease in NO, which contributes to the narrowing of the vascular bed, thrombosis, and activation of VW remodeling mechanisms. Evaluation of ETN-1 levels in NSA patients with different stages of AH showed an increase in ETN-1 concentration parallel to the stage of AH. Peak values of ETN-1 level (15.29 ± 1.4 ng / L with an average confidence level) were detected in NSA in stage III AH (table 2).

Table 2. The content of ETN-1 in the blood of patients with NSA.

Groups	N	The amount of ETN in 1 ng/l
Control	30	4,3 ± 0,58

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NSA + AH II st.	46	12,6 ± 0,97*[1]
NSA + AH III st.	35	15,29 ± 1,4 *[1,2]
Note: *– p<0,05 – statistically significant differences from the control and other groups.		

Table 3. The number of MEC in patients with NSA.

Groups	N	Indicators CEC (cell/100 mkl)
Control	30	4,1 ± 0,6
NSA + AH II ст.	46	9,2 ± 0,5 *[1]
NSA + AH III ст.	35	12,6 ± 0,8 *[1,2]
Note: *– p<0,05 – statistically significant differences from the control and other groups.		

The study of the number of MEC in patients with NSA showed that its highest values were recorded in patients with NSA with arterial hypertension (AH) in stage III (Table 3), and they were statistically significantly higher in contrast to the other two categories of patients examined - NSA with Ar II Art. and control (table. 2).

We have shown a statistically significant increase in the endotheliocythemia parameter in parallel with an increase in the degree of hypertension stage (table. 4).

In NSA, a direct correlation was found between the parameters of TBCIS and markers of endothelial dysfunction (MED) (R = 0.71 with an average confidence level of p <0.01) and an indirect interdependence of indicators of TBCIS and EDVR parameters (r = –0.59 with an average level of confidence).

Table 4. Indicators of blood flow in the brachial artery in patients with NSA.

Indicators	Groups of subjects		
	Control (n=30)	NSA+AH II st. 46)	NSA+AH III st. 35)
Initial Diameter in mm	4,2 ± 0,1	3,8 ± 0,2	3,6 ± 0,2
Diameter of artery for 30 s of reactive eremia, mm	5,1 ± 0,2	4,4 ± 0,1***	4,2 ± 0,1***
The diameter of the artery at 60 with ctive hyperemia, mm	4,8 ± 0,1	4,1 ± 0,1***	3,9 ± 0,1***

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The diameter of the artery at 90 with ctive hyperemia, mm	4,5 ± 0,1	3,9 ± 0,1***	3,7 ± 0,1***
Dilatation caused by flow,%	10,5 ± 2,3	6,2 ± 1,3*	6,0 ± 4,3
Dilatation due to nitroglycerin,%	18,4 ± 2,5	15,7 ± 2,4	15,2 ± 2,5
Initial blood flow rate, ml / s	1,14 ± 0,29	1,06 ± 0,17	1,02 ± 0,4
Measurement of blood flow during ctive hyperemia,%	209,1 ± 36,4	128,2 ± 32,4*	120 ± 27,3*
Note: *– p<0,05; ***– p<0,001 – significant differences from control group data.			

Thus, disproportions of the subpopulation composition of lymphocytes, an increase in the destruction of TBCIS characteristic for different levels of AH, an increase in ETN-1 production, and a decrease in the level of dilatation are observed in NSA patients with differentiated AH levels. These shifts indicate that the NSA is associated with impaired VW and VEW function. A significant place for TBCIS in the pathogenesis of endothelial vascular destruction in NSA can be distinguished.

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