



## MARKERS OF VASCULAR ENDOTHELIAL ACTIVATION AND MARKERS OF INFLAMMATION IN METABOLIC SYNDROME

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### Abstract

Recent advances in medicine and biological sciences have significantly expanded the knowledge of normal proliferation processes and its disorders in pathology [1,2]. Studying the mechanisms of regulation of cell proliferation is extremely important not only in theory, but also in practice, as it allows us to reveal the regularities of growth regulation and identify violations of this process in many diseases [2, 11] and, in particular, in diabetes mellitus (DM) [2] and metabolic syndrome (MS) [4].

**Keywords:** medicine, biological sciences, diabetes mellitus.

### Introduction

MS is characterized by polarization of the immune response, which is manifested by hyperproduction of cytokine-induced cells. At the same time, cytokine-induced overexpression of cell adhesion molecules (MCAS) is noted. In recent years, it has been proposed to consider MCA as an alternative marker of cell-mediated immune activation in a number of diseases, including MS.

One of the markers of inflammation that are informative for assessing the risk of cardiovascular complications in MS patients is C-reactive protein (CRB). It was found that an increase in its level is associated with the risk of complications of coronary heart disease in healthy individuals and is a predictor of complications of coronary heart disease and coronary mortality in patients with coronary heart disease. In recent years, it has been shown that an increase in the level of CRP is combined with a 1.05-2.0-fold increase in the population of circulating endothelial progenitor cells, the release of which is aimed at repairing damaged areas of the vascular wall, which proves the role of inflammation in reactive processes in vascular damage.

Apparently, the mutual influence of various components of the immune inflammation response and the functional state of the endothelium is very complex and multifaceted.

The aim of the study was to compare endothelial activation parameters with markers of the inflammatory process in MS patients.

### Materials and Methods

The study included 80 patients (age 35-60 years, average  $46.7 \pm 2.1$  years), of whom 30 had MS. The duration of the disease ranged from 1 to 12 years. The control group consisted of 20 healthy individuals, comparable in gender and age.

The diagnosis of MS was established according to the criteria of the National Cholesterol Education Program, USA (ADT, 2005) and JIS Criteria (2009). All patients underwent a complete clinical,



biochemical and instrumental study to determine the state of carbohydrate (glucose and fasting insulin) lipid metabolism (total cholesterol-TC, triglycerides-TG, HDL, LDL).

The content of proinflammatory cytokines was determined by the method of solid-phase enzyme-linked immunosorbent assay using the test systems of Cytokine LLC (St. Petersburg, Russia) on an enzyme-linked immunosorbent analyzer "Human" (Germany).

The level of CRP (br-CRP) was determined using the solid-phase tmm enzyme-linked immunosorbent assay by J. Highton and P. Hessain (1984) reagents of Humatex CRP kits.

The concentration of soluble molecules of vascular cell adhesion type 1 (SVCAM-1) was evaluated by solid-phase enzyme-linked immunosorbent assay using test systems Bender from Bender Med Systems (Austria).

The level of circulating desquamated endothelial cells (CDEs) was evaluated by the G method. Hiadovec. (1978).

Statistical processing of the obtained results was carried out using the statistical package Statistica.

## Results and Discussion

During the study, all patients were randomly assigned to 3 groups. I Group I consisted of 25 patients with a diagnosis of arterial hypertension (AH), II-stage II-III, group II – 25 patients with a diagnosis of hypertension of stage II-III + obesity, III group III-30 patients with MS.

Table 1 Content of endothelial activation markers and inflammatory markers in MS patients

Groups	of Cytokines (pkg / ml)		br-CRP m / l	SVCAM-1 ng / ml	CDE Cl / 100 µl	IGF-1 ng/ml
	IL-6	TNF-a				
Healthy (n=20)	20,9±2,1	22,6±2,1	1,22±0,05	645,0±32,3	3,6±0,4	198,6±17,2
Patients with arterial hypertension (n=25)	34,6±2,7	35,2±2,7	3,56±0,022	936,7±51,7	9,5±0,65	247,2±19,6
Patients with arterial hypertension + obesity (n=25)	47,9±3,6	51,2±3,8	4,72±0,39	1131,2±72,3	11,6±0,82	288,4±21,8
Patients with metabolic syndrome (n=30)	83,36,8	101,8±9,6	8,34±0,56	1535,2±84,5	15,7±1,22	324,7±25,3

Inflammatory markers play a special role in the development of MS. In our study, proinflammatory cytokines and CPAs were identified as markers of inflammation. Proinflammatory cytokines play a special role in the induction of vascular damage. They enhance the expression of adhesive molecules, stimulate endothelial procoagulant activity, alter endothelial function, and initiate and maintain chronic inflammation in the arterial walls.

A history of inflammatory markers revealed that the content of interleukin-6 (IL-6) and tumor necrosis factor (TNF-a) in the groups of patients was statistically significant than in the control group (Table 1).1). Statistically significant differences in these parameters were found in all groups of patients. Thus, the maximum level of IL-6 ( $p < 0.001$ ) and TNF-a ( $p < 0.001$ ) was found in MS patients.



## Literature

1. Konnov M. V., Doborjginidze L. M., Deev A.D. et al. Comparison of "old "(2005) and" new " (2009) criteria for assessing metabolic syndrome in families of individuals with premature coronary heart disease. *Cardiology*. 2011;5:4-8.
2. Mammadov M. N., Oganov R. G. Is it necessary to determine insulin resistance for the diagnosis of metabolic syndrome in clinical practice? *Cardiology*. 2005; 4:92-96.
3. Nesterov Yu. I. Poltavtseva O. V., Lazareva O. A. et al. Metabolic syndrome: prevalence among outpatients with arterial hypertension, effectiveness of treatment. *A wedge. the medicine*. 2008;2:67-70.
4. Perova N. V., Ozerova I. N., Alexandrovich O. V. and others. Clinical significance of insulin resistance in fasting normoglycemia. *Cardiology*. 2011; 8:49-53.
5. Shestakova M. V., Butrova S. A., Sukhareva O. Yu. Metabolic syndrome as a harbinger of the development of type 2 diabetes mellitus and cardiovascular diseases. *Ter. archive*. 2007; 10:5-8.
6. American Diabetes Association. Standards of Medical Care in Diabetes – 2010. *Diabetes Care*. 2010; 33 (suppl. 1): 11- 61.
7. Cameron A.J., Magliano D.J., Zimmet P.Z. et al. The metabolic syndrome as a tool for predicting future diabetes: the AusDiab Study. *J. Intern. Med*. 2008; 264: 177-186.
8. Eddi D.M. Schlessinger L. Heikes K. The metabolic syndrome and cardiovascular risk : implication for clinical practice. *Int J Obes (Long)*. 2008; 32 (Suppl 2):S5-10.
9. Ford E.S., Li C., Zhao G. Prevalence and correlates of metabolic syndrome based on a harmonious definition among adults in the US. *J. Diabetes*. 2010; 2: 180-193.
10. Mottillo S., Filion K.B., Genest J. et al. The metabolic syndrome and cardiovascular risk a systematic review and meta – analysis. *J. Am. Col. Cardiol*. 2010; 56: 1113-1132.
11. Reaven G. M. Role of insulin resistance in human disease. *Diabetes*. 1988; 37 (12): 1595-1607.
12. Simmons R.K., Alberli K.G., Gale E.A. et al. The metabolic syndrome useful concept or clinical tool? Report of a WHO Expert Consultation. *Diabetologia*. 2010; 53: 600-605.
13. Doniyorovna, K. Z., & Rakhimovich, B. S. (2022). IMMUNOLOGICAL FEATURES OF INFECTIOUS MONONUCLEOSIS OF EPSTEIN-BARR VIRAL ETIOLOGY IN CHILDREN. *INTERNATIONAL JOURNAL OF SOCIAL SCIENCE & INTERDISCIPLINARY RESEARCH* ISSN: 2277-3630 Impact factor: 7.429, 11, 156-157.
14. Нарзуллаев, Н. У., Мирзаева, М. Р., & Келдиёрова, З. Д. (2020). Цитокиновый Профиль Детей С Острым Воспалением Небных Миндалин При Остром Инфекционном Мононуклеозе На Фоне Лечения. *Новый день в медицине*, (2), 459-461.