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Disaggregation Effects Of Blood Vessels On Neutrophils In Patients With Arterial Hypertension With Hyperuricemia.

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ABSTRACT

A high level of psycho-emotional stress and excess nutrition leads a large part of the population of industrially developed countries to a high incidence of a combination of hypertension and hyperuricemia. At the same time in this category of patients there is a high incidence of thrombosis of different localization. This is caused in these patients by violations of vascular functions, primarily their disaggregation capabilities with respect to blood cells. The goal is to assess the disaggregation effects of blood vessels on neutrophils in patients with hypertension with hyperuricemia. We examined 55 patients of the second mature age (mean age 54.4±2.4 years) with arterial hypertension of the 1st-2nd degree, with hyperuricemia. The control group was composed of 26 clinically healthy people of the same age. All the examined persons gave written informed consent on participation in the research. There were applied biochemical, hematological and statistical methods of investigation.High thromboses' frequency of various localizations at arterial hypertension with hyperuricemia is closely connected with angiopathy development against their background. Weakening of plasma antioxidant protection with activation of lipids' peroxidation processes in it leading to alteration of vascular wall, is noted in conditions of arterial hypertension combination with hyperuricemia. The persons with arterial hypertension and hyperuricemia are detected to have evident weakening of disaggregating vascular impacts of vascular wall on strengthening aggregative ability of neutrophils. In the result of it given patients get sharply increased risk of thromboses of any localization which can lead to invalidism and lethal outcome.

Keywords: neutrophils, arterial hypertension, hyperuricemia, vascular wall, antiaggregation.

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INTRODUCTION

Excess intake of salted, meat foods and high stress levels leads to a high incidence of combination of arterial hypertension (AH) and hyperuricemia among the majority of the population of developed countries [1, 2]. Very often, a combination of this pathology occurs in persons of working age, causing them to have a higher incidence of vascular thrombosis, contributing to disability and early death [3]. It was noted that a high incidence of thrombosis in persons with hypertension and hyperuricemia with a depression of vascular functional activity in terms of their disaggregation capabilities [4,5]. It is known that excessive aggregation of blood elements develops largely due to vascular dysfunction, accompanied by activation of hemostasis and risk of thrombosis [6,7,8]. This process is ensured by the depression of synthesis in the vessels of disaggregants, primarily prostacyclin and nitric oxide [9,10]. The frequent occurrence in the population of a combination of hypertension with hyperuricemia and serious significance for microcirculation of the aggregation capacity of neutrophils, it was important to assess the state of vascular disaggregation control over them in this category of patients [11].

The aim of the study is to assess the disaggregation effects of vessels on neutrophils in hypertensive patients with hyperuricemia.

MATERIALS AND METHODS

The research was approved by the Ethics Committee of Russian State Social University (record №5 from 12.05.2014).

We examined 41 patients of the second mature age (mean age 54.4±2.4 years) with AH of the 1^{st}-2^{nd} degree [12] with hyperuricemia. The control group was composed of 26 clinically healthy people of the same age. All the examined persons gave written informed consent on participation in the research. All participants in the study gave their written consent to participate in it [13].

Intensity of lipids' peroxidation (LPO) processes in plasma was estimated according to the content of thiobarbituric acid (TBA)-active products by a kit "Agat-Med" and acylhydroperoxides (AHP) [14]. Antioxidant abilities of liquid part of blood were determined according to the level of its antioxidant activity [15].

LPO activity in studied regular blood elements was determined according to the quantity of malondialdehyde (MDA) in reduction reaction of thiobarbituric acid in washed and resuspended cells and the content of AHP in them [14]. In studied washed and resuspended regular blood elements we estimated the levels of cholesterol by enzymatic colorimetric method with the help of a kit "Vital Diagnostikum" and CPL according to the content of phosphorus in them.

Evidence of vascular wall's control over neutrophils' aggregation was detected according to its weakening in the test with temporal venous occlusion [16].

Vessel control over neutrophil aggregation was assessed in plasma taken under conditions of temporary venous occlusion and without it on a photoelectric colorimeter. Inductors were used lectin wheat germ at a dose of 32 µg / ml, concanavalin A - 32 µg / ml and phytohemagglutinin - 32 µg / ml.

The results were processed by Student's criterion (t). Statistical processing of received information was made with the help of a program package "Statistics for Windows v. 6.0", "Microsoft Excel". Differences in data were considered reliable in case of $p<0.05$.

RESULTS AND DISCUSSION

The patients were noted to have evident plasma LPO activation – the content of AHP in it surpassed the control value in 2.3 times, TBA-active products – in 1.5 times, being accompanied by suppression of antioxidant plasma activity in 1.5 times (Table).

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The observed patients were noted to have increased CS content in neutrophils membranes which was accompanied by the decrease of CPL in them and LPO activation on behalf of weakening of their antioxidant protection (Table).

All the patients were noted to have the decrease of vessels' disaggregative impacts on neutrophils (Table).

Table: Registered indicators in the surveyed

Registrated parameters	Patients, n=41, M±m	Control, n=26, M±m
acylhydroperoxides plasma, D_{233} /1ml	3.27±0.08	1.42±0.09 p<0.01
TBA-compounds, mcmol / l	5.38±0.12	3.56±0.07 p<0,01
antioxidantactivityplasma, %	22.2±0.17	32.9±0.12 p<0.01
biochemical parameters of neutrophils		
cholesterol of neutrophils, mkmol/10^9 neutrophils	0.85±0.012	0.62±0.004 p<0.01
common phospholipids of neutrophils, mkmol/10^9 neutrophils	0.34±0.006	0.51±0.003 p<0.01
acylhydroperoxides of neutrophils, D_{233}/10^9 neutrophils	3.68±0.08	2.36±0.05 p<0.01
malonicdialdehyde of neutrophils, nmol/10^9 neutrophils	1.53±0.09	0.73±0.03 p<0.01
catalase of neutrophils, ME/10^9 neutrophils	5300.0±21.85	9950.0±19.77 p<0.01
superoxidismutase of neutrophils, ME/10^9 neutrophils	1250.0±4.17	1780.0±4.21 p<0.01
aggregation of neutrophils in intact plasma		
Aggregationwithlectin, %	24.2±0.15	15.6±0.07 p<0.01
Aggregation withconcanavalin A, %	21.0±0.12	14.8±0.04 p<0.01
Aggregationwithphytohemagglutinin, %	42.2±0.08	30.6±0.09 p<0.01
vascular control of aggregation neutrophils		
Aggregationwithlectinafter temporary venous occlusion, %	20.9±0.15	11.8±0.06 p<0.01
Aggregation withconcanavalin Aafter temporary venous occlusion, %	19.2±0.09	11.0±0.07 p<0.01
Aggregationwithphytohemagglutininafter temporary venous occlusion, %	39.2±0.21	24.1±0.03 p<0.01

Note: p - reliability of differences in the indices of a group of patients and a control group.

In plasma, taken against the background of temporary venous occlusion, the patients showed redundancy of neutrophil aggregation exceeding the values of the control group with all applied inducers (from lectin on 77.1%, from concanavalinA on74.5%, from phytohemagglutinin on 62.6%).

Important significance in the development of rheological disturbances and thrombophilia in persons with AH and hyperuricemia belongs to aggregation increase of regular blood elements and especially – neutrophils [17,18]. At combination of AH and hyperuricemia the depression of plasma antioxidant activity is formed which provides the increase of LPO activity in it [19]. The increase of freely radical processes in liquid part of blood inevitably promotes the damage of neutrophils' membranes [20]. The development of these

manifestations in combination with found in these patients' neutrophils lipid imbalance leads to their hyperaggregability. The level of disaggregating impacts from the side of vascular wall [21,22] lowers simultaneously with it in respect of neutrophils [23].

The increase in neutrophil aggregation in patients enrolled in the study is strongly associated with a weakened synthesis in the vascular wall of desaggregants, while the glycoprotein receptor activity on the surface of leukocytes increases with respect to lectins used as inducers [24,25]. The amplification caused by lectin and concanavalin A of neutrophil aggregation in plasma taken against a background of temporary venous occlusion in hypertensive patients with hyperuricemia is associated with an increase in the expression on the membrane of neutrophil receptors, which include in their composition many sites including N-acetyl-D-glucosamine, N -acetyl-neuraminic acid and mannose [26, 27]. Redundancy of neutrophil aggregation in response to phytohemagglutinin is associated with an increase in the area of their receptors containing bD-galactose [28,29] with a weakened synthesis in the vessels of patients with prostacyclin and NO [30,31,32].

CONCLUSION

The high frequency of occurrence in a modern society of a combination of arterial hypertension with hyperuricemia requires a detailed study of this pathology. Great attention to it is caused by a high incidence of thrombosis in this category of patients. In the conducted research it was established that in these patients in the plasma lipid peroxidation processes were intensified. Apparently, it is they that cause the development of vasopathy with a weakening of the production in the vessels of antiplatelet agents. This leads to a weakening in patients with vascular control over increasing aggregation of neutrophils. The weakening of the disaggregation capacity of the vessels and the increased aggregation of neutrophils reduce the trophism of tissues and increase the risk of thrombosis in hypertensive patients with hyperuricemia.

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