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Vascular Disaggregation Control Of Neutrophils In Patients With Arterial Hypertension With Impaired Glucose Tolerance.

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ABSTRACT

Despite the efforts of medicine among the population of industrially developed countries, arterial hypertension and violation of glucose tolerance are very widespread. It is recognized that the high prevalence of thromboses in arterial hypertension and impaired glucose tolerance is associated with a weakening of the vascular functions and, in the first place, their disaggregation effects on the blood elements. The goal is to clarify the disaggregation capabilities of the vessels in relation to neutrophils in patients with arterial hypertension with impaired glucose tolerance. We examined 49 patients of the second mature age (mean age 52.4±1.9 years) with arterial hypertension of the 1^{st}-2^{nd} degree, with impaired glucose tolerance. 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 impaired glucose tolerance 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 impaired glucose tolerance. The persons with arterial hypertension and impaired glucose tolerance 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, impaired glucose tolerance, vascular wall, antiaggregation.

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INTRODUCTION

Despite the increased attention to the level of health of the population in industrially developed countries, the prevalence of a combination of arterial hypertension (AH) and impaired glucose tolerance is preserved [1,2]. Very often, their combination develops in able-bodied individuals, causing them a high incidence of vascular complications leading to disability and early mortality [3]. It becomes clear that a high frequency in the population of thromboses in hypertension and impaired glucose tolerance is associated with a weakening of the synthetic processes in the vessels, especially their disaggregation control factors over the shaped elements [4,5]. It is recognized that the strengthening of the aggregation of blood elements occurs necessarily in vascular dysfunctions, accompanied by activation of hemostasis and the development of thrombosis [6-8]. This is largely due to a decrease in synthesis in the vessels of disaggregants, including prostacyclin and nitric oxide [9,10]. In view of the high prevalence of hypertension with impaired glucose tolerance and serious significance for microcirculation of neutrophils, it was important to assess the level of vascular control over the process of aggregation of neutrophilic leukocytes in these patients [11].

The aim of the work is to clarify the disaggregation capabilities of the vessels in relation to neutrophils in patients with hypertension with a tolerance to glucose tolerance.

MATERIALS AND METHODS

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

We examined 55 patients of the second mature age (mean age 51.2±2.7 years) with AH of the 1^{st}-2^{nd} degree [12] withimpaired glucose tolerance. 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].

The effect of vessels on neutrophil aggregation was assessed in plasma taken after temporary venous occlusion and without it on a photoelectric colorimeter. Inductors were the lectin of wheat germ at a concentration of 32 µg / ml, concanavalin A - 32 µg / ml and phytohemagglutinin - 32 µg / ml.

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 1.43 times (Table).

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).

In the observed patients, neutrophil aggregation in response to applied inductors appeared earlier than in the control group (with lectin at 48.7%, concanavalin A 35.8%, with phytohemagglutinin 34.9%) (Table).

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In all patients, a decrease in the disaggregation effects of the vessels with respect to neutrophils (Table).

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Table: Registered indicators in the surveyed

Registrated parameters	Patients, n=49, M±m	Control, n=26, M±m
acylhydroperoxides plasma, D_{233} /1ml	3.25±0.08	1.42±0.09 p<0.01
TBA-compounds, mcmol / l	5.27±0.15	3.56±0.07 p<0,01
antioxidantactivityplasma, %	23.0±0.18	32.9±0.12 p<0.01
biochemical parameters of neutrophils		
cholesterol of neutrophils, mkmol/10^9 neutrophils	0.85±0.010	0.62±0.004 p<0.01
common phospholipids of neutrophils, mkmol/10^9 neutrophils	0.36±0.009	0.51±0.003 p<0.01
acylhydroperoxides of neutrophils, D_{233}/10^9 neutrophils	3.62±0.04	2.36±0.05 p<0.01
malonicdialdehyde of neutrophils, nmol/10^9 neutrophils	1.46±0.05	0.73±0.03 p<0.01
catalase of neutrophils, ME/10^9 neutrophils	5150.0±12.09	9950.0±19.77 p<0.01
superoxidismutase of neutrophils, ME/10^9 neutrophils	1200.0±4.12	1780.0±4.21 p<0.01
aggregation of neutrophils in intact plasma		
Aggregationwithlectin, %	23.2±0.16	15.6±0.07 p<0.01
Aggregation withconcanavalin A, %	20.1±0.13	14.8±0.04 p<0.01
Aggregationwithphytohemagglutinin, %	41.3±0.06	30.6±0.09 p<0.01
vascular control of aggregation neutrophils		
Aggregationwithlectinafter temporary venous occlusion, %	20.6±0.19	11.8±0.06 p<0.01
Aggregation withconcanavalin Aafter temporary venous occlusion, %	18.0±0.05	11.0±0.07 p<0.01
Aggregationwithphytohemagglutininafter temporary venous occlusion, %	39.2±0.18	24.1±0.03 p<0.01

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

Important significance in the development of rheological disturbances and thrombophilia in persons with AH and impaired glucose tolerance belongs to aggregation increase of regular blood elements and especially – neutrophils [17,18]. At combination of AH and impaired glucose tolerance 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 intensification of neutrophil aggregation detected in the examined patients is largely due to the depression of synthesis in the vessel walls of compounds having disaggregation activity against the background of an increase in the number of glycoprotein receptors on leukocytes to lectins used as inducers in the study [24,25]. The intensification of lectin- and concanavalin A-induced aggregation of neutrophils in plasma after temporary venous occlusion in patients with hypertension with impaired glucose tolerance is associated with an increase in the expression level on the membranes of their neutrophilic adhesion receptors, which contain a significant number of sites containing N-acetyl-D -glucosamine, N-acetyl-neuraminic acid and mannose [26, 27]. The enhancement of neutrophil aggregation in response to the appearance of phytohemagglutinin in the plasma is caused by the growth in their receptors of sites of glycoproteins containing bD-galactose [28,29] under the conditions of depression of synthesis in the vascular endothelium of prostacyclin and NO patients [30,31,32].

CONCLUSION

The frequent occurrence in modern people of a combination of arterial hypertension with a violation of glucose tolerance requires a comprehensive study of this pathology. Particular attention to neutrophils is due to the high incidence of thrombosis in this category of patients. In the study, it was found that lipid peroxidation in plasma was significantly enhanced in these patients. This contributes to the formation of vasopathy with a weakening of vascular production of physiological antiplatelet agents. This weakens their vascular control over the dramatically increasing aggregation of neutrophils. The simultaneous weakening of the disaggregation properties of blood vessels and the enhancement of neutrophil aggregation disrupt trophism of tissues and make a significant contribution to the risk of thrombosis in patients with arterial hypertension with impaired glucose tolerance.

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