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The Ability To Aggregate Neutrophils In Patients With Dyslipidemia.

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ABSTRACT

The excessive consumption of food in modern society is accompanied by the growth in the population of many industrially developed countries of dyslipidemia. With this pathology, many doctors associate the frequent occurrence of thrombosis of various localizations. In their basis, the hyperaggregation of the blood cells is likely to occur after dyslipidemia. The goal is to establish the characteristics of neutrophil aggregation in patients with dyslipidemia. We examined 41 patients of the second adult age (mean age 53.8 ± 1.6 years) with dyslipidemia type IIb. The control group consisted of 26 clinically healthy people of the same age. All the examined persons gave written informed consent to participate in the study. Biochemical, hematological and statistical methods of investigation were used. The high frequency of thromboses of different localizations, characteristic for this category, is largely related to the hyperaggregation of neutrophils. A serious reason for enhancing the ability to aggregate in neutrophilic leukocytes is the weakening of the antioxidant defense of the plasma with the activation of the processes of lipid peroxidation in it. In the work carried out, it was found that people with hypertension and dyslipidemia have a weakening of neutrophil disaggregation. As a result, patients receive a sharply increased risk of thrombosis of any location, which can lead to disability and death.

Keywords: neutrophils, pathology, dislipidemia, vascular wall, antiaggregation.

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INTRODUCTION

Excess caloric content and excessive fat content in food provide an improvement in the population of industrially developed countries the prevalence of dyslipidemia [1,2]. Its presence provides for a working part of the population a high incidence of vascular thrombosis, leading to disability and early death [3]. Many researchers associate the frequent occurrence of thromboses in dyslipidemia with the development of hyperaggregation of blood cells [4,5]. It is known that hyperaggregation of blood elements leads to the initiation of hemostasis and thrombosis [6,7,8]. This process is largely caused by a decrease in the sensitivity of blood cells to vascular disaggregants, including prostacyclin and nitric oxide [9,10]. Given the widespread prevalence of dyslipidemia and greater significance for microcirculation of neutrophils, it seemed important to assess the level of aggregation of neutrophilic leukocytes in this contingent of patients [eleven].

The goal is to establish the features of neutrophil aggregation in patients with dyslipidemia.

MATERIAL AND METHODS

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

We examined 41 patients of the second mature age (mean age 53.8±1.6 years) with dislipidemia of IIb type [12]. 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.

We determined the content of common cholesterol (CS) and triglycerides (TG) in blood of all the observed persons by enzymatic colorimetric method with the help of a kit “Vital Diagnostikum” (Russia). CS level of high-density lipoproteins (HDLP) was determined with the help of a kit “Olveks Diagnostikum (Russia) by enzymatic colorimetric method. Common lipids (CL) were estimated with the help of a kit “Erba Russ” (Russia). The quantity of common phospholipids (CPL) in blood plasma was registered according to the content of phosphorus in them. CS levels of low-density lipoproteins (LDLP) were established by calculation according to Freedwald V. CS concentrations of very low-density lipoproteins (VLDLP) was determined according to the formula: TG content/2.2. Received indices of common CS and CS of LDLP were considered as normal, border-line or high in accordance with Russian recommendations (2012) [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 (AOA) [15].

LPO activity in studied regular blood elements was determined according to the quantity of malon dialdehyde (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.

The level of neutrophil aggregation was assessed on a photoelectrocolorimeter [16]. As inductors, a wheat germ lectin was used 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 $p<0.05$.

RESEARCH RESULTS AND DISCUSSION

The blood of patients was noted to have levels’ increase of CL and common CS which surpassed the control values in 1.6 and 1.3 times, respectively, at simultaneous lowering of plasma CPL in 2.1 times (Table). The blood of persons with dislipidemia was found to have the increase of CS LDLP, CS VLDLP and TG in 1.64, 1.61 and 1.60 times, respectively. It is combined with the lowering of CS HDLP in 1.5 times. The patients were

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noted to have evident plasma LPO activation – the content of AHP in it surpassed the control value in 2.1 times, TBA-active products – in 1.4 times, being accompanied by suppression of antioxidant plasma activity in 1.3 times (Table).

Table. Registered indicators in the surveyed

Registrated parameters	Patients with arterial hypertension and dyslipidemia, n=41, M±m	Control, n=26, M±m
common cholesterol, mmol / l	6.3±0.05	4.8±0.05 p<0.01
CS level of high-density lipoproteins, mmol /l	1.07±0.06	1.60±0.06 p<0.01
CS levels of low-density lipoproteins, mmol /l	3.99±0.07	2.43±0.04 p<0.01
CS concentrations of very low-density lipoproteins, mmol /l	1.24±0.04	0.77±0.05 p<0.01
triglycerides, mmol /l	2.72±0.06	1.70±0.02 p<0.01
common lipids, g/l	9.0±0.10	5.6±0.03 p<0.01
common phospholipids, mmol/l	1.70±0.06	3.54±0.09 p<0.01
acylhydroperoxides plasma, D_{233} /1ml	3.01±0.07	1.42±0.09 p<0.01
TBA-compounds, µmol/l	4.92±0.08	3.56±0.07 p<0,01
antioxidant activity plasma, %	24.2±0.13	32.9±0.12 p<0.01
biochemical parameters of neutrophils		
cholesterol of neutrophils, µmol/10^9 neutrophils	0.80±0.007	0.62±0.004 p<0.01
common phospholipids of neutrophils, µmol /10^9 neutrophils	0.39±0.003	0.51±0.003 p<0.01
acylhydroperoxides of neutrophils, D_{233}/10^9 neutrophils	3.36±0.07	2.36±0.05 p<0.01
malonic dialdehyde of neutrophils, nmol/10^9 neutrophils	1.37±0.06	0.73±0.03 p<0.01
catalase of neutrophils, ME/10^9 neutrophils	5600.0±18.05	9950.0±19.77 p<0.01
superoxidismutase of neutrophils, ME/10^9 neutrophils	1300.0±5.01	1780.0±4.21 p<0.01
aggregation of neutrophils		
Aggregation with lectin, %	21.3±0.07	15.6±0.07 p<0.01
Aggregation with concanavalin A, %	18.4±0.12	14.8±0.04 p<0.01
Aggregation with phytohemagglutinin, %	40.6±0.07	30.6±0.09 p<0.01

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

The observed patients were noted to have increased CS content in erythrocytes' 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 patients enrolled, neutrophil aggregation with all tested inducers appeared earlier than in the control (with lectin at 36.5%, concanavalin A at 24.3%, with phytohemagglutinin 32.7%) (Table).

Important significance in the development of rheological disturbances and thrombophilia in persons with dislipidemia belongs to aggregation increase of regular blood elements and especially – neutrophils [17,18]. At dislipidemia 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. At the same time, the level of platelets decreases with platelets [21,22, 23].

The increase in neutrophil aggregation in patients under observation appears to be largely due to a drop in their sensitivity to disaggregants against the background of negative reorganizations of glycoprotein receptors of leukocytes to lectins used as inductors [24,25]. The intensification of lectin- and concanavalin A-induced neutrophil aggregation in patients with dyslipidemia was caused by an increase in expression on the membrane by neutrophil receptor adhesion with an increase in their composition of sites containing N-acetyl-D-glucosamine, N-acetyl-neuraminic acid and mannose [26, 27]. The redundancy of neutrophil aggregation induced by phytohemagglutinin aggregation is attributed to the authors attributed to the growth of glycoproteins containing bD-galactose [28,29] in their receptors with decreasing blood levels in patients with prostacyclin and NO [30,31,32].

CONCLUSION

The high frequency of thrombosis of various localizations with dyslipidemia is closely related to the development of excessive aggregation of neutrophils. An important reason for this is the weakening of the antioxidant protection of the plasma with the activation of lipid peroxidation processes in it. The study found that people with dyslipidemia have a weakening of the ability to disaggregate in neutrophils. As a result, patients with dyslipidemia receive a sharply increased risk of thrombosis of any location, which can lead to disability and death [33,34,35].

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