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## Antiaggregational Vascular Control Of Erythrocytes In Patients With Dyslipidemia.

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### ABSTRACT

The high incidence of vascular thrombosis in patients with dyslipidemia is largely due to the presence of vasopathy in them. Given the widespread prevalence of dyslipidemia in modern society, it is of great scientific and practical interest to assess the state of these patients in vascular control of aggregation by the most numerous group of blood cells - red blood cells. The aim of the work is to establish the features of the disaggregation effects of blood vessels on erythrocytes in patients with dyslipidemia. 41 patients with dyslipidemia IIb of the second adult age were under observation. The control group consisted of 26 clinically healthy volunteers of the same age. Biochemical, hematological and statistical methods of investigation have been applied. In the examined patients, an increase in the cholesterol erythrocyte membranes and decrease in total phospholipids in them and activation of lipid peroxidation was found. Increased spontaneous aggregation of erythrocytes was found in all the examined patients. In this case, all patients noted a decrease in vascular control over erythrocyte aggregation. The pronounced weakening of disaggregating vascular control over the enhanced aggregation capacity of red blood cells is a consequence of the dyslipidemia-induced vasopathy. As a result, in patients with dyslipidemia, the risk of thrombosis of any location increases, which can lead to disability and death.

**Keywords:** dyslipidemia, vascular wall, antiaggregation, erythrocytes, thrombosis risk.

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## INTRODUCTION

Large-scale preventive measures so far cannot contain the spread among the population of industrially developed countries of dyslipidemia [1,2]. This pathology is accompanied by a high incidence of vascular complications, very often leading to disability and mortality [3,4].

The high incidence of vascular thrombosis in patients with dyslipidemia is largely due to the development of vasopathy [5,6]. It is recognized that blood cells are capable of aggregation, which leads to activation of hemostasis and development of thrombosis [7,8,9]. These processes are constrained by the synthesis in the wall of the vessel and the release of them into the blood of the disaggregants. The most important of these are prostacyclin and nitric oxide [10,11]. Due to the widespread prevalence of dyslipidemia, it is of great scientific and practical interest to continue research into the features of vascular control over the aggregation of the most abundant blood cells - red blood cells in this patient population.

The goal is to establish the features of the disaggregation effects of blood vessels on erythrocytes in patients with dyslipidemia.

## MATERIALS AND METHODS

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

41 patients with dyslipidemia IIb type [12], second adult age (mean age  $53.8 \pm 1.6$  years) were examined. The control group consisted of 26 clinically healthy people of similar age. All surveyed gave written information consent to participate in the study.

In the work by the enzymatic colorimetric method, the total blood cholesterol (CH) and triglycerides (TG) were determined by the "Vital Diagnosticum" kit (Russia). The level of high-density lipoprotein (HDL) cholesterol was determined using the OlvexDiagnosticum (Russia) assay using the enzymatic colorimetric method. The total lipids (OL) were assessed by the Erba Russ kit (Russia), the total amount of phospholipids (OPL) of the blood plasma was recorded from the content of phosphorus in them. The levels of low-density lipoprotein (LDL) cholesterol were established by the formula of Friedwald B. The concentration of lipoprotein cholesterol was very low density (VLDL) was determined by the formula: the content of TG/2.2. The results of total cholesterol and LDL cholesterol were considered normal, borderline or high in accordance with the Russian recommendations (2012) [13].

Levels of lipid peroxidation (LPO) in plasma were evaluated by the content of thiobarbituric acid (TBA)-active products with the Agat-Med kit and acyl hydroperoxides (AHP). [14] The antioxidant capacity of the liquid part of the blood was determined from the level of its antioxidant activity (AOA) [15].

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

## RESULTS AND DISCUSSION

In the examined patients, an increase in the levels of OL and total cholesterol exceeding the control values of 1.6 and 1.3 times, respectively, was revealed, respectively. This was accompanied by a fall in their plasma OPL by 2.1 times (Table 1). In the blood of individuals with dyslipidemia, an increase in LDL cholesterol, LLDPE cholesterol and TG was found in 1.64, 1.61, and 1.60 times, respectively. At the same time, they had a 1.5-fold decrease in HDL cholesterol. Also, the activation of plasma LPL was revealed in patients - the content of AGP in it exceeded the control by 2.1 times, TBA-active products - 1.4 times. This was due to the weakening of the antioxidant activity of the plasma by a factor of 1.3 (Table).

**Table: Patient survey results**

<b>Registered parameters</b>	<b>Patients with dyslipidemia, n=41, M±m</b>	<b>Control, n=26, M±m</b>
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 <sub>233</sub> /1ml	3.01±0.07	1.42±0.09 p<0.01
TBA-compounds, mcmol/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
<b>biochemical parameters of erythrocytes</b>		
cholesterol of erythrocytes, mkmol/10 <sup>12</sup> erythrocytes	1.32±0.005	1.04±0.004 p<0.01
common phospholipids of erythrocytes, mkmol/10 <sup>12</sup> erythrocytes	0.56±0.006	0.75±0.003 p<0.01
acylhydroperoxides of erythrocytes, D <sub>233</sub> /10 <sup>12</sup> erythrocytes	4.42±0.12	3.08±0.10 p<0.01
malonic dialdehyde of erythrocytes, nmol/10 <sup>12</sup> erythrocytes	1.60±0.14	1.14±0.05 p<0.01
catalase of erythrocytes, ME/10 <sup>12</sup> erythrocytes	7680.0±13.7	11196.0±22.4 p<0.01
superoxidismutase of erythrocytes, ME/10 <sup>12</sup> erythrocytes	1620.0±1.95	1986.0±7.01 p<0.01
<b>aggregation of erythrocytes in intact plasma</b>		
sum of all the erythrocytes in an aggregate	67.3±0.10	41.9±0.10 p<0.01
quantity of aggregates	12.8±0.13	9.0±0.06 p<0.01
quantity of free erythrocytes	167.3±1.52	240.0±0.23 p<0.01
<b>aggregation of erythrocytes in plasma after temporary venous occlusion</b>		
sum of all the erythrocytes in an aggregate	55.2±0.10	32.6±0.14 p<0.01
quantity of aggregates	10.0±0.14	7.0±0.07 p<0.01
quantity of free erythrocytes	201.3±1.06	305.3±0.18 p<0.01

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

Observed patients noted an excess of cholesterol in the erythrocyte membranes, accompanied by a decrease in them in OPL, activation of LPO and weakening of their antioxidant protection (Table).

In the blood of patients with dyslipidemia, a marked increase in spontaneous aggregation of erythrocytes was recorded (Table). This was indicated by an increased level of their total involvement in aggregates (by 60.6%), an increase in the number of aggregates themselves (by 42.2%) and a decrease of 43.4% in freely moving red blood cells.

In patients with dyslipidemia, there was a decrease in the disaggregation effects of blood vessels in relation to erythrocytes (Table). It was found that in the plasma obtained against the background of temporary venous occlusion, the total number of erythrocytes in aggregates in the aggregates exceeded the control by 69.3%, the number of these aggregates was increased by 42.8%, accompanied by a decrease in the number of free red blood cells by 51.7%.

An important role in the development of rheological disorders and thrombophilia in individuals with dyslipidemia belongs to the intensification of aggregation of blood cells and especially erythrocytes [18, 19]. With dyslipidemia, there is a depression of the antioxidant activity of the plasma, which increases the activity of LPO in it [20]. The increase in free-radical processes in the plasma inevitably contributes to damage to erythrocyte membranes [21]. The development of these phenomena in combination with the lipid imbalance found in the erythrocytes of these patients leads to their hyperaggregability. At the same time, the level of disaggregating effects from the vessels decreases in relation to erythrocytes [22,23]. This proved in the examined patients an increase in the aggregation activity of erythrocytes in plasma obtained after a temporary venous occlusion [24]. Increased spontaneous aggregation of erythrocytes in vivo in patients with dyslipidemia is caused by a weakening of the disaggregating effects of the vessels [25,26] and the developing decrease in the number of negatively charged proteins on the outer erythrocyte membrane [27]. Weakening of antioxidant activity of plasma leads to an increase in the level of active forms of oxygen in it and to oxidative damage of endotheliocytes, erythrocyte membrane proteins and plasma proteins [28,29]. With a clear deficit of physiological disaggregants, the binding of erythrocytes to each other in the already formed aggregates is enhanced [30, 31]. The weakening of production in the vessels of prostacyclin and nitric oxide leads to an imbalance in the erythrocytes of the activity of adenylatecyclase and phosphodiesterase [32,33]. Under these conditions, the amount of cyclic adenosine monophosphate decreases in their cytoplasm and the level of Ca<sup>2+</sup> increases, further strengthening the aggregation of erythrocytes [34, 35].

## CONCLUSION

The wide prevalence of thrombosis of various localizations with dyslipidemia is caused by the development of vasopathy on its background. In the presence of dyslipidemia in patients, weakened antioxidant protection of the plasma with the activation of LPO processes in it, leading to alteration of the vascular wall. For persons with dyslipidemia, the disaggregating vascular effects of the vascular wall are weakened by the aggregative capacity of erythrocytes, which increases in these conditions. For this reason, these patients have an increased risk of vessel thrombosis of any location, which can lead to disability and death [36,37,38].

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