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## Aggregational Properties Of Erythrocytes In Patients With Dyslipidemia.

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### ABSTRACT

The wide prevalence of thrombosis in patients with dyslipidemia is largely due to the presence of hyperaggregating blood cells in them. Given the widespread prevalence of dyslipidemia among the population, it is of great interest to assess the state of this pathology of aggregation of the most numerous group of red blood cells. The aim of this work is to establish the activity of erythrocyte aggregation in patients with dyslipidemia. Under observation, 41 patients with dyslipidemia IIb of the second adult age were taken. 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 cholesterol and a decrease in total phospholipids in the activation of lipid peroxidation were found in erythrocyte membranes. Increased spontaneous aggregation of erythrocytes was found in all the examined patients. The pronounced increase in the aggregating properties of erythrocytes is a consequence of metabolic disturbances that arise during dyslipidemia. As a result, in patients with dyslipidemia, the risk of thrombosis of any location increases, which can lead to disability and death.

**Keywords:** dyslipidemia, pathology, aggregation, erythrocytes, thrombosis risk.

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## INTRODUCTION

Planned medical preventive measures in different countries have not yet been able to contain the spread of dyslipidemia among the population [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 hyperaggregation of their blood cells [5,6]. It is recognized that this process leads to activation of hemostasis and development of thrombosis [7,8,9]. He comes very actively when their ability to disaggregate weakens. 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 numerous red blood cells - the erythrocytes in this contingent of patients.

The goal is to establish the activity of erythrocyte aggregation in patients with dyslipidemia.

## MATERIAL 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 and triglycerides were determined by the "Vital Diagnosticum" kit (Russia). The level of high-density lipoprotein cholesterol was determined using the Olvex Diagnosticum (Russia) assay using the enzymatic colorimetric method. The total lipids were assessed by the Erba Russ kit (Russia), the total amount of phospholipids of the blood plasma was recorded from the content of phosphorus in them. The levels of low-density lipoprotein cholesterol were established by the formula of Friedwald B. The concentration of lipoprotein cholesterol was very low density was determined by the formula: the content of triglycerides/2.2 The results of total cholesterol and cholesterol low density lipoprotein 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 [15].

The expression of lipid peroxidation in erythrocytes was determined from the amount of malonic dialdehyde (MDA) in the reduction reaction of thiobarbituric acid after washing and resuspension and the content of AHP in them [14]. In washed and resuspended erythrocytes, cholesterol levels were evaluated by an enzymatic colorimetric method using the "Vital Diagnosticum" and total phospholipids-containing phosphorus.

The state of aggregation of erythrocytes was determined with the help of a light microscope in Goryaev's chamber [16], recording the number of erythrocyte aggregates, the number of aggregates and erythrocytes not aggregated [17].

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  $p < 0.05$ .

## RESEARCH RESULTS AND DISCUSSION

The examined patients showed an increase in the levels of total lipids and total cholesterol exceeding the control values by 1.6 and 1.3 times, respectively. This was accompanied by a decrease in their total plasma

phospholipids by a factor of 2.1 (Table 1). In the blood of individuals with dyslipidemia, an increase in low-density lipoprotein cholesterol, low-density lipoprotein cholesterol and a triglyceride of 1.64, 1.61 and 1.60 times, respectively, was found. In this case, they have a decrease in the cholesterol of high-density lipoproteins by a factor of 1.5. Also, the activation of plasma LPL was revealed in patients - the content of AHP 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**

Registered parameters	Patients with 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 <sub>233</sub> /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 erythrocytes		
cholesterol of erythrocytes, µmol/10 <sup>12</sup> erythrocytes	1.32±0.005	1.04±0.004 p<0.01
common phospholipids of erythrocytes, µmol/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
aggregation of erythrocytes		
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

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 erythrocyte membranes, accompanied by a decrease in total phospholipids in them, activation of LPO, and weakening of their antioxidant defense (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.

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. In this case, the level of their disaggregating properties in the erythrocytes weakens [22,23]. This was proved in the examined patients by the stability of the formed aggregates of erythrocytes [24]. The increase in spontaneous aggregation of erythrocytes in vivo in patients with dyslipidemia is caused not only by the weakening of their disaggregating properties [25,26], but also by 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]. It becomes clear that with dyslipidemia, there is an increase in the binding of erythrocytes among themselves in already formed aggregates [30, 31]. The weakening of their sensitivity to disaggregants causes an imbalance in the erythrocytes of the activity of adenylate cyclase and phosphodiesterase [32,33]. Under these conditions, the amount of cyclic adenosine monophosphate decreases in their cytoplasm and the level of  $Ca^{2+}$  increases. This additionally enhances the aggregation of erythrocytes [34,35].

#### CONCLUSION

The wide prevalence of thrombosis of various localizations in dyslipidemia is caused by the development of hyperaggregation of blood cells against its background. In the presence of dyslipidemia in patients, weakened antioxidant protection of the plasma with the activation of LPO processes leading to the alteration of erythrocyte membranes, which enhances their aggregation ability. For this reason, these patients have a high risk of vascular thrombosis of any location, which can lead to disability and death [36,37,38].

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