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Expression Of Aggregation Capacity Of Platelets In Abdominal Obesity And Dyslipidemia.

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ABSTRACT

The gradual steady improvement of the process of rendering medical care to the population can not contain the growth in the world of prevalence of a combination of abdominal obesity and dyslipidemia. A particular danger of this condition is the high incidence of thrombosis development. This circumstance is commonly associated with hyperaggregation of blood cells, which has not been studied to the end. The goal is to assess the level of aggregation properties of platelets in patients with abdominal obesity and dyslipidemia. We examined 41 patients of the second adult age (mean age 49.2 ± 1.8 years) with abdominal obesity and dyslipidemia. The control group consisted of 26 clinically healthy people of the same age. All the examinees gave written informed consent to participate in the study. Biochemical, hematological and statistical methods of investigation were used. The high incidence of thrombosis of various localizations with abdominal obesity and dyslipidemia is closely related to the development of angiopathy against their background. Weakening of antioxidant protection of blood plasma with the activation of lipid peroxidation processes that lead to a change in the vascular wall is noted in conditions of abdominal obesity and dyslipidemia. In individuals with abdominal obesity and dyslipidemia, a pronounced weakening of the disaggregating vascular effects of the vascular wall was shown to increase the aggregative capacity of thrombocytes. In individuals with abdominal obesity and dyslipidemia, a pronounced weakening of the disaggregating vascular effects of the vascular wall was shown to increase the aggregative capacity of thrombocytes. As a result of his patients, the risk of thrombosis of any localization increases sharply, which can lead to disability and death.

Keywords: platelets, abdominal obesity, dyslipidemia, aggregation, hemostasis.

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INTRODUCTION

Recently, a high prevalence of a combination of abdominal obesity and dyslipidemia has been preserved [1,2]. In patients with this combination, very often vascular thrombosis develops leading to disability, and sometimes to death [3]. This is often based on hyperaggregation of blood cells [4]. This inevitably leads to activation of hemostasis and creates conditions favoring thrombosis [5,6,7]. Activation of the aggregation of blood cells is often accompanied by a weakening of their sensitivity to vascular disaggregants - prostacyclin and nitrogen oxide [8,9]. Given the prevalence of abdominal obesity and dyslipidemia, it seemed important to evaluate platelet aggregation in patients with this pathology [10].

The goal is to assess the level of aggregation properties of platelets in patients with abdominal obesity and dyslipidemia.

MATERIAL 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 49.2±1.8 years) with abdominal obesity and dyslipidemia [11]. 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 those surveyed agreed to participate in the study [12].

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) [13]. Antioxidant abilities of liquid part of blood were determined according to the level of its antioxidant activity [14].

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 [13]. 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 total phospholipids according to the content of phosphorus in them.

The activity of platelet aggregation (AP) in the study was assessed using a visual micromethode [15] using ADP ($0.5x10^{-4}$ M), collagen (1: 2 dilution of the base suspension), thrombin (0.125 U/ml), ristomycin (0.8 mg/ml), epinephrine (5.0×10^{-6} M) and with combinations of ADP and epinephrine; ADP and collagen; adrenaline and collagen at the same plasma concentrations standardized for platelet count to 200×10^{9} platelets/l [16]. Aggregational properties of platelets inside the vessels were determined using a phase contrast microscope, taking into account the number of small, medium and large aggregates and the degree of platelet involvement in them in plasma [17,18].

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 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).

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

In the examined patients with abdominal obesity and dyslipidemia, acceleration of AP with inductors and their combinations was found (Table). In the past, AP occurred in response to collagen, a little later on

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ADP, even later on ristomycin, thrombin and adrenaline. AP in response to a combination of inducers also developed accelerated. The number of circulating patients with platelet aggregates and the degree of platelet involvement in those with abdominal obesity and dyslipidemia significantly exceeded the level of the control group.

Table. Registered indicators in the surveyed

Registrated parameters	Patients, n=41, M±m	Control, n=26, M±m
acylhydroperoxides plasma, D_{233} /1ml	3.29±0.08	1.42±0.09 p<0.01
TBA-compounds, µmol / l	5.38±0.09	3.56±0.07 p<0,01
antioxidant activity plasma, %	21.8±0.23	32.9±0.12 p<0.01
biochemical parameters of platelets		
cholesterol of platelets, mkmol/10^9 platelets	1.08±0.007	0,67±0,005 p<0,01
common phospholipids of platelets, mkmol/10^9 platelets	0.34±0.012	0,49±0,004 p<0,01
acylhydroperoxides of platelets, D_{233}/10^9 platelets	3.42±0.10	2,20±0,04 p<0,01
malonic dialdehyde of platelets, nmol/10^9 platelets	1.41±0.12	0,68±0,02 p<0,01
catalase of platelets, ME/10^9 platelets	5600.0±25.82	9790,0±20,10 p<0,01
superoxidismutase of platelets, ME/10^9 platelets	1200.0±10.17	1650,0±3,00 p<0,01
aggregation of platelets		
aggregation with ADP, s	25.2±0.12	41,0±0,12 p<0,01
aggregation with collagen, s	23.8±0.16	33,2±0,10 p<0,01
aggregation with thrombin, s	37.4±0.13	55,3±0,05 p<0,01
aggregation with ristomycin, s	28.5±0.14	45,2±0,06 p<0,01
aggregation with epinephrine, s	71.4±0.20	93,0±0,07 p<0,01
aggregation with ADP and epinephrine, s	21.8±0.25	34,5±0,04 p<0,01
aggregation with ADP and collagen, s	18.7±0.16	26,6±0,05 p<0,01
aggregation with epinephrine and collagen, s	13.5±0.18	29,2±0,12 p<0,01
The number of platelets in the aggregates, %	10.2±0.15	6,5±0,07 p<0,01
Number of little aggregates (in 100 free thrombocytes)	13.8±0.20	3,1±0,03 p<0,01
Number of medium and large aggregates (in 100 free thrombocytes)	1.62±0.07	0,14±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 abdominal obesity and dyslipidemia belongs to aggregation increase of regular blood elements and especially – platelets [19,20]. At combination of abdominal obesity and dyslipidemia the depression of plasma antioxidant activity is formed which provides the increase of LPO activity in it [21,22]. The increase of freely radical processes in liquid part of blood inevitably promotes the damage of platelets' membranes. The development of these manifestations in combination with found in these patients' platelets lipid imbalance leads to their hyperaggregability. At the same time, platelets lower the level of disaggregating abilities [23,24].

Reduction of the sensitivity of platelets to vascular disaggregants is caused by simultaneous enhancement of mechanisms and the weakening of platelet disaggregation mechanisms [25,26]. Apparently, a serious cause of this can be the activation of LPO in plasma [27,28]. Previously, the development of AP with ristomycin in patients should be associated with increased synthesis in the walls of their vascular Willebrand factor [29,30]. The accelerated onset of AP in response to the appearance in the environment of a combination of inducers and an excessive number of platelet aggregates in the blood in patients is a consequence of the resulting weakening of the disaggregation properties of platelets [31, 32].

CONCLUSION

The aggregative properties of platelets are an important element in ensuring the liquid state of the blood. With the development of thrombocytopathy, it is inevitable that in patients the attenuation of their disaggregation possibilities begins. Very often it occurs in metabolic disorders, especially with abdominal obesity and dyslipidemia. The high incidence of this pathology is the reason for evaluating the aggregation capacity of platelets in this contingent of patients. It was found out that in the case of abdominal obesity and dyslipidemia in patients, a marked increase in aggregation capacity of platelets is recorded. These manifestations should be considered as a serious cause of activation in such patients of hemostasis mechanisms and formation of a high risk of thrombosis [33,34,35].

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