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Activity Of Platelet Aggregation In Patients With Impaired Glucose Tolerance And Abdominal Obesity.

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ABSTRACT

Despite the serious success of medicine, the prevalence of a combination of abdominal obesity and impaired glucose tolerance persists in developed countries. This category of patients is very threatened by the development of thromboses caused by the presence of hyperaggregation of blood cells in them, the characteristics of which have not yet been fully investigated. The goal is to find out the aggregation capacity of platelets in patients with abdominal obesity with impaired glucose tolerance. 39 patients of the second adult age (mean age 50.1 ± 1.8 years) with violation of glucose tolerance and abdominal obesity were examined. 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 in the work. It became clear that a high incidence of thrombosis of various localizations in abdominal obesity with impaired glucose tolerance is closely related to the development of platelet hyperaggregation. An important reason for its development should be considered the weakening of antioxidant protection of blood plasma with the activation of lipid peroxidation in it. In individuals with abdominal obesity and impaired glucose tolerance, a pronounced weakening of the disaggregatory properties of platelets was revealed. As a result of his patients, the risk of thrombosis of any localization increases sharply, which can lead to disability and death.

Key words: platelets, abdominal obesity, impaired glucose tolerance, thrombophilia, aggregation.

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INTRODUCTION

Despite the serious efforts of medicine, the prevalence of a combination of abdominal obesity and impaired glucose tolerance remains high in the world [1,2]. The presence of this combined pathology in patients causes a high risk of vascular thrombosis, dangerous disability and premature death [3]. An important mechanism for the risk of thrombosis of any location in these patients is hyperaggregation of blood cells [4]. Its formation is accompanied by a weakening of the sensitivity of blood cells to vascular disaggregation control, which strongly stimulates the mechanisms of hemostasis [5,6,7]. Functionally, it is very important to reduce the sensitivity of blood cells to prostacyclin and nitric oxide [8,9]. In view of the prevalence of the combination of abdominal obesity and impaired glucose tolerance, it was important to evaluate the state of platelet aggregation in this category of patients [10].

The goal is to clarify the aggregation capacity of platelets in patients with abdominal obesity with impaired glucose tolerance.

MATERIAL AND METHODS

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

We examined 39 patients of the second mature age (mean age 50.1 ± 1.8 years) with impaired glucose tolerance and abdominal obesity [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 severity of platelet aggregation (AP) was assessed using a visual micromethode [16] using ADP (0.5×10^{-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; epinephrine and collagen at the same concentrations. The study was conducted in a platelet-rich plasma with a standardized platelet count of 200×10^9 platelets. Intravascular aggregation of thrombocytes was determined using a phase contrast microscope with the registration of the number of small, medium and large aggregates and the degree of involvement of platelets in them [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 2.2 times, TBA-active products – in 1.4 times, being accompanied by suppression of antioxidant plasma activity in 1.32 times (Table).

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

Table. Registered indicators in the surveyed

Registered parameters	Patients, n=39, M±m	Control, n=26, M±m
acylhydroperoxides plasma, $D_{233}/1\text{ml}$	3.10 ± 0.06	1.42 ± 0.09 $p < 0.01$
TBA-compounds, $\mu\text{mol/l}$	5.08 ± 0.12	3.56 ± 0.07 $p < 0.01$
antioxidant activity plasma, %	24.8 ± 0.17	32.9 ± 0.12 $p < 0.01$
biochemical parameters of platelets		
cholesterol of platelets, $\mu\text{mol}/10^9 \text{ platelets}$	1.01 ± 0.016	0.67 ± 0.005 $p < 0.01$
common phospholipids of platelets, $\mu\text{mol}/10^9 \text{ platelets}$	0.39 ± 0.005	0.49 ± 0.004 $p < 0.01$
acylhydroperoxides of platelets, $D_{233}/10^9$ platelets	3.20 ± 0.09	2.20 ± 0.04 $p < 0.01$
malonic dialdehyde of platelets, $\text{nmol}/10^9$ platelets	1.21 ± 0.10	0.68 ± 0.02 $p < 0.01$
catalase of platelets, $\text{ME}/10^9 \text{ platelets}$	5600.0 ± 26.18	9790.0 ± 20.10 $p < 0.01$
superoxidismutase of platelets, $\text{ME}/10^9$ platelets	1200.0 ± 8.73	1650.0 ± 3.00 $p < 0.01$
aggregation of platelets		
aggregation with ADP, s	27.4 ± 0.14	41.0 ± 0.12 $p < 0.01$
aggregation with collagen, s	28.2 ± 0.15	33.2 ± 0.10 $p < 0.01$
aggregation with thrombin, s	39.4 ± 0.08	55.3 ± 0.05 $p < 0.01$
aggregation with ristomycin, s	31.0 ± 0.05	45.2 ± 0.06 $p < 0.01$
aggregation with epinephrine, s	75.4 ± 0.12	93.0 ± 0.07 $p < 0.01$
aggregation with ADP and epinephrine, s	23.6 ± 0.12	34.5 ± 0.04 $p < 0.01$
aggregation with ADP and collagen, s	19.5 ± 0.09	26.6 ± 0.05 $p < 0.01$
aggregation with epinephrine and collagen, s	15.8 ± 0.16	29.2 ± 0.12 $p < 0.01$
The number of platelets in the aggregates, %	10.1 ± 0.15	6.5 ± 0.07 $p < 0.01$
Number of little aggregates (in 100 free thrombocytes)	12.3 ± 0.17	3.1 ± 0.03 $p < 0.01$
Number of medium and large aggregates (in 100 free thrombocytes)	1.39 ± 0.09	0.14 ± 0.03 $p < 0.01$

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

In patients with abdominal obesity and impaired glucose tolerance, a pronounced acceleration of development of AP with inductors and their combinations was found (Table). The most accelerated AP developed with collagen, a little later with ADP, even later with ristomycin, thrombin and adrenaline. The onset of AP with combinations of inductors occurred even more rapidly. The number of platelet aggregates and the level of platelet involvement in patients with abdominal obesity and impaired glucose tolerance were significantly higher than the control numbers.

Important significance in the development of rheological disturbances and thrombophilia in persons with abdominal obesity and impaired glucose tolerance belongs to aggregation increase of regular blood elements and especially – platelets [19,20]. At combination of abdominal obesity and impaired glucose tolerance 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 reduced the ability to disaggregate [23,24].

Amplification of AP with inductors and their combinations is caused not only by strengthening of AT mechanisms, but also by weakening mechanisms of disaggregation [25,26]. A very important role in this is the activation of LPO in plasma [27,28]. Acceleration of the course of the process of AP with ristomycin in patients is associated with an increase in the number of receptors on platelets to the factor of von Willebrand [29,30]. Early development in patients with AP in response to combinations of two inducers and the presence of a large number of platelet aggregates in their blood is a consequence of a strong depression of their ability to disaggregate [31, 32].

CONCLUSION

Aggregational properties of platelets are extremely important for maintaining homeostasis in the body. An important element of its violation is their hyperaggregation. This condition is now very common in patients with any metabolic pathology, including with abdominal obesity and impaired glucose tolerance. The high incidence of this pathology dictates the need for a detailed assessment of the aggregation capacity of platelets in this patient population. It was found that with abdominal obesity in violation of glucose tolerance there is a pronounced weakening of the platelets ability to disaggregate. These manifestations in this contingent of patients are a serious cause of activation of hemostasis and the formation of a risk of developing thrombosis of any localization [33,34,35].

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