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Aggregation Of Platelets In Patients With Impaired Glucose Tolerance.

Medvedev IN*.

Russian State Social University, st. V. Pika, 4, Moscow, Russia, 129226.

ABSTRACT

For quite a long time researchers have noted an increase in the number of patients suffering from impaired glucose tolerance in the world. At the same time, they register a high incidence of thrombosis, which is associated with hyperaggregation of blood cells, many aspects of which are still not sufficiently investigated. The goal is to evaluate the aggregation properties of platelets in patients with impaired glucose tolerance. To achieve this goal, 42 patients of the second adulthood (mean age 49.6±1.7 years) with impaired glucose tolerance 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. It became clear that a high incidence of thrombosis of various localizations in violation of glucose tolerance is closely related to the development of excessive aggregation of platelets against their background. This was largely due to the weakening of the antioxidant defense of blood plasma with the activation of lipid peroxidation processes in it. Also, in individuals with impaired glucose tolerance, a pronounced weakening of the ability to disaggregate their platelets was found. As a result of his patients, the risk of thrombosis of any localization increases sharply, which can lead to disability and death.

Keywords: platelets, impaired glucose tolerance, thrombophilia , aggregation.

Corresponding author

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INTRODUCTION

In previous studies, it was firmly established that the prevalence of impaired glucose tolerance persists in industrially developed countries [1,2]. This is accompanied by a greater risk of developing in these patients vascular thrombosis, dangerous disability and death [3]. It is believed that the emergence of thrombosis of any localization, including in these patients, promotes hyperaggregation of blood cells [4]. Its formation is manifested by intensification of the process of aggregation of all blood elements, which stimulates all mechanisms of hemostasis [5,6,7]. Against the background of hyperaggregation of blood elements, there is also a marked weakening of their sensitivity to desagregant substances, the most functionally significant of which are prostacyclin and nitric oxide [8,9]. In view of the high prevalence of impaired glucose tolerance, it was of great interest to evaluate the state of platelet aggregation in these patients [10].

The goal is to evaluate the aggregation properties of platelets in patients 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 42 patients of the second mature age (mean age 49.6±1.7 years) with impaired glucose tolerance [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.

Platelet aggregation activity (AP) was assessed using a visual micromethode [15] in plasma obtained without and using venous occlusion 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; epinephrine and collagen at the same concentrations in the platelet-rich plasma as standardized for the platelet count of 200×10^{9} platelets. The severity of platelet aggregation capacity in intravascular conditions 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 2.1 times, TBA-active products – in 1.4 times, being accompanied by suppression of antioxidant plasma activity in 1.25 times (Table).

The observed patients were noted to have increased cholesterol 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).

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In patients with impaired glucose tolerance, 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 was even more accelerated. The number of platelet aggregates and the level of involvement of platelets in patients with impaired glucose tolerance exceeded the control figures.

Table. Registered indicators in the surveyed

Registrated parameters	Patients, n=42, M±m	Control, n=26, M±m
acylhydroperoxides plasma, D_{233} /1ml	2.93±0.07	1.42±0.09 p<0.01
TBA-compounds, µmol/l	4.87±0.12	3.56±0.07 p<0,01
antioxidant activity plasma, %	26.2±0.16	32.9±0.12 p<0.01
biochemical parameters of platelets		
cholesterol of platelets, µmol/10^9 platelets	1.04±0.012	0,67±0,005 p<0,01
common phospholipids of platelets, µmol/10^9 platelets	0.37±0.006	0,49±0,004 p<0,01
acylhydroperoxides of platelets, D_{233}/10^9 platelets	3.25±0.07	2,20±0,04 p<0,01
malonic dialdehyde of platelets, nmol/10^9 platelets	1.24±0.19	0,68±0,02 p<0,01
catalase of platelets, ME/10^9 platelets	5750.0±20.54	9790,0±20,10 p<0,01
superoxidismutase of platelets, ME/10^9 platelets	1250.0±8.62	1650,0±3,00 p<0,01
aggregation of platelets		
aggregation with ADP, s	28.5±0.16	41,0±0,12 p<0,01
aggregation with collagen, s	27.6±0.14	33,2±0,10 p<0,01
aggregation with thrombin, s	39.4±0.12	55,3±0,05 p<0,01
aggregation with ristomycin, s	31.4±0.04	45,2±0,06 p<0,01
aggregation with epinephrine, s	78.5±0.17	93,0±0,07 p<0,01
aggregation with ADP and epinephrine, s	23.8±0.19	34,5±0,04 p<0,01
aggregation with ADP and collagen, s	19.6±0.11	26,6±0,05 p<0,01
aggregation with epinephrine and collagen, s	16.7±0.09	29,2±0,12 p<0,01
The number of platelets in the aggregates, %	10.3±0.10	6,5±0,07 p<0,01
Number of little aggregates (in 100 free thrombocytes)	11.4±0.14	3,1±0,03 p<0,01
Number of medium and large aggregates (in 100 free thrombocytes)	1.46±0.06	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 impaired glucose tolerance belongs to aggregation increase of regular blood elements and especially – platelets [19,20]. At 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 increased the level of aggregation capabilities [23,24].

Acceleration of AP with inductors and their combinations is caused not only by strengthening of AP mechanisms, but also by weakening mechanisms of disaggregation [25,26]. Apparently, an important role in this is the activation of LPO in plasma [27,28]. Acceleration of the process of AP with ristomycin in patients is associated with an increase in the sensitivity of platelets to the factor of von Willebrand and an increase in its content in their plasma [29,30]. A rapid onset 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 activation of their aggregation mechanisms in the blood [31, 32].

CONCLUSION

Aggregational activity of thrombocytes is an important indicator of the preservation of homeostasis in the body. A serious manifestation of its disorders is an increase in the aggregation capacity of thrombocytes. This phenomenon is very common in many variants of pathology, including the violation of glucose tolerance. Its wide prevalence dictates the need to evaluate the aggregation capacity of platelets in this contingent of patients. These disorders in this contingent of patients are a serious cause of activation of hemostasis and the development of a risk of thrombosis of any localization [33,34,35].

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