

Research Journal of Pharmaceutical, Biological and Chemical Sciences

Platelet Aggregation Activity In Patients With Abdominal Obesity.

Medvedev IN*.

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

ABSTRACT

Among the adult population of the world, there is still a high incidence of abdominal obesity. These patients have seen a high incidence of thrombosis, which they associate with the development of hyperaggregation in their blood cells. The goal is to evaluate the aggregation capacity of platelets in patients with abdominal obesity. We examined 46 patients of the second adulthood (mean age 52.7 ± 2.2 years) with abdominal obesity. The control group consisted of 26 clinically healthy people of the same age. All the examined persons 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 in abdominal obesity is closely related to the development of platelet hyperaggregation in these patients. An important reason for the formation of this situation should be considered the weakening of antioxidant protection of the plasma with activation of the processes of lipid peroxidation in it. It was found that people with abdominal obesity have an obvious weakening of platelet disaggregation. As a result, patients receive a sharply increased risk of thrombosis of any location, which can lead to disability and death.

Keywords: platelets, abdominal obesity, vascular wall, antiaggregation.

**Corresponding author*

INTRODUCTION

Improving the quality of life in a modern civilized society is accompanied by a high incidence of abdominal obesity [1,2]. This fact causes a high incidence of development in adults of the mass of vascular thrombosis leading to widespread disability and mortality [3]. A serious cause of high frequency of thrombosis of any location in any contingent of patients is almost always excessive aggregation of blood cells, the prevalence of which is now increasing [4]. As a result, the mechanisms of hemostasis are stimulated and the risk of thrombosis increases [5,6,7]. During development, there is a weakening of the sensitivity of blood cells to disaggregants, of which the most important are prostacyclin and nitric oxide [8,9]. In view of the prevalence of abdominal obesity, it seemed very important to study the state of platelet aggregation in this patient population [10].

The goal is to assess the aggregation capacity of platelets in patients with abdominal obesity.

MATERIAL AND METHODS

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

We examined 46 patients of the second mature age (mean age 52.7 ± 2.2 years) with 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 the total phospholipids according to the content of phosphorus in them.

The activity of platelet aggregation (AP) was elucidated by visual micromethod [15] in plasma obtained without and with venous occlusion using ADP (0.5×10^{-4} M), collagen (1: 2 dilution of the basic 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 a platelet-rich plasma with a standardized platelet count of 200×10^9 platelets [16]. Intravascular aggregation of platelets was determined using a phase contrast microscope in terms of the number of small, medium and large aggregates and the 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 2.0 times, TBA-active products – in 1.3 times, being accompanied by suppression of antioxidant plasma activity in 1.3 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).

In individuals with abdominal obesity, acceleration of development of AP with inductors and their combinations was revealed (Table). Most quickly, AP came with collagen, a little later with ADP, even later with ristomycin, thrombin and epinephrine. The development of AP with combinations of inductors was also accelerated. The number of free platelet aggregates in the blood and the level of platelet involvement in them in individuals with abdominal obesity exceeded control figures.

Table. Registered indicators in the surveyed

Registered parameters	Patients, n=46, M±m	Control, n=26, M±m
acylhydroperoxides plasma, D ₂₃₃ /1ml	2.92±0.08	1.42±0.09 p<0.01
TBA-compounds, mcmol/l	4.85±0.12	3.56±0.07 p<0,01
antioxidant activity plasma, %	25.0±0.16	32.9±0.12 p<0.01
biochemical parameters of platelets		
cholesterol of platelets, mkmol/10 ⁹ platelets	0.92±0.007	0,67±0,005 p<0,01
common phospholipids of platelets, mkmol/10 ⁹ platelets	0.36±0.005	0,49±0,004 p<0,01
acylhydroperoxides of platelets, D ₂₃₃ /10 ⁹ platelets	3.08±0.05	2,20±0,04 p<0,01
malonic dialdehyde of platelets, nmol/10 ⁹ platelets	1.10±0.08	0,68±0,02 p<0,01
catalase of platelets, ME/10 ⁹ platelets	6310.0±17.21	9790,0±20,10 p<0,01
superoxidismutase of platelets, ME/10 ⁹ platelets	1280.0±7.23	1650,0±3,00 p<0,01
aggregation of platelets		
aggregation with ADP, s	29.9±0.14	41,0±0,12 p<0,01
aggregation with collagen, s	27.9±0.12	33,2±0,10 p<0,01
aggregation with thrombin, s	42.4±0.10	55,3±0,05 p<0,01
aggregation with ristomycin, s	32.3±0.07	45,2±0,06 p<0,01
aggregation with epinephrine, s	77.2±0.15	93,0±0,07 p<0,01
aggregation with ADP and epinephrine, s	26.0±0.10	34,5±0,04 p<0,01
aggregation with ADP and collagen, s	19.8±0.08	26,6±0,05 p<0,01
aggregation with epinephrine and collagen, s	21.3±0.10	29,2±0,12 p<0,01
The number of platelets in the aggregates, %	10.1±0.12	6,5±0,07 p<0,01
Number of little aggregates (in 100 free thrombocytes)	11.4±0.15	3,1±0,03 p<0,01
Number of medium and large aggregates (in 100 free thrombocytes)	1.02±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 belongs to aggregation increase of regular blood elements and especially – platelets [19,20]. At combination of abdominal obesity 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 [23,24].

Activation of AP with inductors and their combinations is caused by the simultaneous enhancement of AP mechanisms and the weakening of mechanisms of disaggregation [25,26]. Apparently, an important reason for this is the activation of plasma LPO [27,28]. Acceleration of AP with ristomycin in patients is caused by increased synthesis in the walls of the vessels of von Willebrand factor [29,30]. Rapid development of AP in response to a combination of inducers and a large number of platelet aggregates in patients is a consequence of the pronounced weakening of platelet disaggregation properties [31, 32].

CONCLUSION

Platelet homeostasis is an important component of maintaining the health of the body. One of the manifestations of its disorders is an increase in aggregation properties of platelets. This phenomenon is very common in metabolic disorders, including abdominal obesity. The high prevalence of abdominal obesity in society dictated the need to evaluate the aggregation properties of platelets in this contingent of patients. It was revealed that abdominal obesity showed a marked increase in platelet aggregation. These disorders in the examined patients were a serious basis for activation of hemostasis processes and development of thromboses of any localization in them [33,34,35].

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