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Increased Aggregation Properties Of Platelets In Patients With Hyperuricemia.

Medvedev IN*.

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

ABSTRACT

The development of preventive measures of modern medicine so far does not allow reducing the number of patients who have hyperuricemia. Great attention to this category of patients is associated with the risk of developing in them thrombosis of vessels of any localization. The leading reason for this is the presence of hyperaggregation of blood cells in them, the mechanisms of which have not yet been fully clarified. The aim is to evaluate the aggregation properties of platelets in patients with hyperuricemia. We examined 32 patients of the second adulthood (mean age 51.3 ± 2.3 years) with hyperuricemia. The control group consisted of 26 clinically healthy people of the same age. All examined persons gave written informed consent to participate in the study. Biochemical, hematological and statistical methods of investigation were used. The frequency of high thrombosis of various localizations in hyperuricemia is closely related to the development of angiopathy on their background. Weakening of antioxidant protection of the plasma with activation of the processes of lipid peroxidation in it, leading to a change in the vascular wall, is noted in hyperuricemia. It was found that persons with hyperuricemia have an obvious weakening of the disaggregation of vascular lesions of the vascular wall with an increase in the aggregation capacity of thrombocytes. As a result, patients receive a sharply increased risk of thrombosis of any location, which can lead to disability and death.

Keywords: platelets, vasopathy, hyperuricemia, vascular wall, antiaggregation.

Corresponding author

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INTRODUCTION

Continuous development of medicine has so far failed to contain the spread among the population of the mature age of developed countries of hyperuricemia [1,2]. This disease is very dangerous development of vascular thrombosis with it [3]. For their high frequency, these patients always have hyperaggregation of blood cells, the prevalence of which also does not decrease [4]. The resulting excess aggregation of blood cells strongly stimulates hemostasis and is an important cause of the risk of thrombosis [5,6,7]. It is known that hyperaggregation is always manifested by depression of the sensitivity of blood cells to disaggregants, especially to prostacyclin and nitric oxide [8,9]. Given the widespread prevalence of hyperuricemia, it seemed important for medical practice to assess the state of platelet aggregation in this contingent of patients [10].

The goal is to evaluate the aggregation properties of platelets in patients with hyperuricemia.

MATERIAL AND METHODS

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

We examined 32 patients of the second mature age (mean age 51.3±2.3 years) with hyperuricemia [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 total phospholipids according to the content of phosphorus in them.

The level of platelet aggregation (AP) was evaluated by the micro-method [15] in plasma obtained without and with venous occlusion in response to ADP ($0.5x10^{-4}$ M), collagen (dilution 1:2 of the base suspension), thrombin (0.125 U/ml), ristomycin (0.8 mg/ml), epinephrine (5.0×10^{-6} M) and with a combination of ADP and epinephrine; ADP and collagen; epinephrine and collagen at the same concentrations in a platelet-rich plasma standardized for platelet counts of 200×10^{9} platelets [16]. Intravascular aggregation of platelets was determined using a phase contrast microscope. Considered in the plasma 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 2.1 times, TBA-active products – in 1.4 times, being accompanied by suppression of antioxidant plasma activity in 1.3 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).

In persons with hyperuricemia, an earlier onset of AP with inductors and their combinations was revealed (Table). Previously, AP was attacked with collagen, a little later with ADP, even later with ristomycin,

thrombin and adrenaline. The onset of AP with combinations of inductors was also accelerated. The number of platelet aggregates and the level of platelet involvement in them in people with hyperuricemia exceeded those of the control group.

Table. Registered indicators in the surveyed

Registrated parameters	Patients, n=32, M±m	Control, n=26, M±m
acylhydroperoxides plasma, D_{233} /1ml	3.06±0.14	1.42±0.09 p<0.01
TBA-compounds, µmol/l	5.01±0.18	3.56±0.07 p<0,01
antioxidant activity plasma, %	24.5±0.15	32.9±0.12 p<0.01
biochemical parameters of platelets		
cholesterol of platelets, µmol/10^9 platelets	1.02±0.002	0.67±0.005 p<0.01
common phospholipids of platelets, µmol/10^9 platelets	0.36±0.006	0.49±0.004 p<0.01
acylhydroperoxides of platelets, D_{233}/10^9 platelets	3.17±0.07	2.20±0.04 p<0.01
malonic dialdehyde of platelets, nmol/10^9 platelets	1.22±0.10	0.68±0.02 p<0.01
catalase of platelets, ME/10^9 platelets	5850.0±20.71	9790.0±20.10 p<0.01
superoxidismutase of platelets, ME/10^9 platelets	1250.0±8.23	1650.0±3.00 p<0.01
aggregation of platelets		
aggregation with ADP, s	28.1±0.17	41.0±0.12 p<0.01
aggregation with collagen, s	26.4±0.18	33.2±0.10 p<0.01
aggregation with thrombin, s	39.7±0.17	55.3±0.05 p<0.01
aggregation with ristomycin, s	31.2±0.19	45.2±0.06 p<0.01
aggregation with epinephrine, s	71.3±0.16	93.0±0.07 p<0.01
aggregation with ADP and epinephrine, s	24.7±0.23	34.5±0.04 p<0.01
aggregation with ADP and collagen, s	19.4±0.16	26.6±0.05 p<0.01
aggregation with epinephrine and collagen, s	16.9±0.25	29.2±0.12 p<0.01
The number of platelets in the aggregates, %	10.3±0.14	6.5±0.07 p<0.01
Number of little aggregates (in 100 free thrombocytes)	14.1±0.15	3.1±0.03 p<0.01
Number of medium and large aggregates (in 100 free thrombocytes)	1.40±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 hyperuricemia belongs to aggregation increase of regular blood elements and especially – platelets [19,20]. At hyperuricemia 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 weaken their disaggregating capabilities [23,24].

Depression of AP with individual inducers and their combinations is associated with the enhancement of AP mechanisms and the weakening of mechanisms of disaggregation [25,26]. A serious reason for this is the activation of LPO in plasma [27,28]. Acceleration of AP in response to ristomycin in patients is also associated with increased synthesis in vascular wall of von Willebrand factor [29,30]. The accelerated onset of AP in response to combinations of inducers and an excess of platelet aggregates in patients is the result of a pronounced weakening of platelet disaggregation mechanisms [31, 32].

CONCLUSION

Increasing the aggregation of blood cells is an important component of thrombophilia. With various pathological processes, it can be amplified, which is manifested, among other things, in platelets. These disorders are very common in metabolic disorders, including hyperuricemia. Its wide distribution among the population prompted the author to evaluate the aggregation properties of platelets in this contingent of patients. It was found that hyperuricemia is characterized by a marked increase in platelet aggregation. This situation seriously violates the hemostatic balance in the body and forms the risk of thrombosis of any localization [33,34,35].

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