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Weakening Of Disaggregation Control Of Blood Vessels Over Platelets In Patients With Hyperuricemia.

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ABSTRACT

Progressive development of modern medicine and continuous preventive measures among the population so far do not allow reducing the number of patients who have hyperuricemia. This category of patients is very threatening for the development of their thrombosis vessels of any localization. The leading reason for this is the presence of vasopathy in them, the nature of which has not yet been clarified. The goal is to assess the level of disaggregation properties of blood vessels on platelets in patients with hyperuricemia. We examined 32 patients of the second mature age (mean age 51.3±2.3 years) with hyperuricemia. 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. There were applied biochemical, hematological and statistical methods of investigation. High thromboses' frequency of various localizations at hyperuricemia is closely connected with angiopathy development against their background. Weakening of plasma antioxidant protection with activation of lipids' peroxidation processes in it leading to alteration of vascular wall, is noted in conditions of hyperuricemia. The persons with hyperuricemia are detected to have evident weakening of disaggregating vascular impacts of vascular wall on strengthening aggregative ability of platelets. In the result of it given patients get sharply increased risk of thromboses of any localization which can lead to invalidism and lethal outcome.

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

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INTRODUCTION

Continuous development of medical science and practice can not yet contain the spread among the population of the mature age of developed countries of hyperuricemia [1,2]. This disease is very dangerous high frequency of development of vascular thrombosis with it [3]. For their high frequency, these patients always have vasopathy, the prevalence of which also does not decrease [4]. Always vasopathy is manifested by the weakening of vascular control over the aggregation of blood cells, which stimulates hemostasis and leads to thrombosis [5,6,7]. It is known that vasopathy is always manifested by the depression of synthesis in the walls of the vessels of disaggregants, especially prostacyclin and nitric oxide [8,9]. Given the widespread prevalence of hyperuricemia, it seemed important for science and practice to assess the state of vascular control of platelet aggregation in this patient population [10].

The goal is to assess the level of disaggregation properties of blood vessels on platelets in patients with hyperuricemia.

MATERIALS 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 CPL according to the content of phosphorus in them.

Evidence of vascular wall's control over platelets' aggregation was detected according to its weakening in the test with temporal venous occlusion [15].

The level of platelet aggregation (AP) was evaluated by the micro-method [16] in plasma obtained without and with venous occlusion in response to ADP ($0.5x10^{-4}$ M), collagen (dilution 1: 2 of the basic suspension), thrombin (0.125 ed/ml), ristomycin (0.8 mg/ml), epinephrine (5.0×10^{-6} M) and with a combination of ADP and epinephrine; ADP and collagen; adrenaline and collagen at the same concentrations in a platelet-rich plasma standardized for platelet counts of 200×10^9 platelets. The value of the index of antiaggregatory activity of the vascular wall (IAASC) was calculated in the course of dividing the duration of AP in plasma after venous occlusion by the duration of it in intact plasma. The disaggregation effects of the vessel wall on intravascular aggregation of platelets were determined using a phase contrast microscope. The number of small, medium, and large aggregates and the involvement of platelets in plasma taken without temporal venous occlusion and in plasma obtained on its background were taken into account [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 $p<0.05$.

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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).

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, umol/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, umol/10^9 platelets	1.02±0.002	0.67±0.005 p<0.01
common phospholipids of platelets, umol/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 in intact plasma		
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

cardiovascular control of platelet aggregation		
IAAVW with ADP	1.28±0.12	1.53±0.16 p<0.01
IAAVW with collagen	1.23±0.20	1.48±0.16 p<0.01
IAAVW with thrombin	1.24±0.17	1.44±0.13 p<0.01
IAAVW with ristomycin	1.29±0.09	1.56±0.11 p<0.01
IAAVW with epinephrine	1.36±0.20	1.62±0.13 p<0.01
IAAVW with ADP and epinephrine	1.32±0.24	1.49±0.12 p<0.01
IAAVW with ADP and collagen	1.28±0.17	1.51±0.10 p<0.01
IAAVW with epinephrine and collagen	1.24±0.14	1.53±0.11 p<0.01
The number of platelets in the aggregates after temporary venous occlusion, %	8.3±0.03	4.5±0.15 p<0.01
Number of little aggregates (in 100 free thrombocytes) after temporary venous occlusion	6.3±0.12	2.1±0.15 p<0.01
Number of medium and large aggregates (in 100 free thrombocytes) after temporary venous occlusion	0.17±0.006	0.02±0.005 p<0.01

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

The observed patients were noted to have increased CS content in erythrocytes' membranes which was accompanied by the decrease of CPL 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.

All the patients were noted to have the decrease of vessels' disaggregative impacts on platelets (Table).

In patients with hyperuricemia, a decrease in IAAWW was found with individual inducers (for adrenaline 1.36±.0.20, for ADP 1.28±0.12, for ristomycin 1.29±0.09, for collagen and thrombin 1.23±0.20 and 1.24±0.17, respectively) and with their combinations (for ADP and adrenaline 1.32±0.24, for ADP and collagen – 1.28±0.17, for adrenaline and collagen – 1.24±0.14). In plasma obtained with temporary venous occlusion, the number of platelet aggregates of any different sizes in the blood of patients and the high involvement of platelets in them decreased only slightly.

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. The level of disaggregating impacts from the side of vascular wall [23,24] lowers simultaneously with it in respect of platelets.

Depression IAAVW with inducers and their combinations is associated with a simultaneous increase in AP and a weakening of the disaggregation capacity of the vessels [25,26]. A serious reason for this is the activation of LPO in plasma [27,28]. Acceleration of AT 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 in the blood taken without venous occlusion and against it is a consequence of the pronounced weakening of the disaggregation capacity of the vascular endothelium [31, 32].

CONCLUSION

Synthesis of disaggregants in the walls of blood vessels is an important component of maintaining the liquid state of the blood. With various pathological processes, it can weaken, which is manifested, among other things, by depression of the disaggregation effects of blood vessels on platelets. These disorders are very common in metabolic disorders, including hyperuricemia. Its wide distribution among the population prompted the author to assess the disaggregation effects on platelets in this contingent of patients. In the work it was revealed that for hyperuricemia there is a pronounced weakening of the disaggregation effects of blood vessels on platelets. 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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