ISSN: 0975-8585

Research Journal of Pharmaceutical, Biological and Chemical Sciences

Aggregational Properties Of Platelets In Patients With Arterial Hypertension With Hyperuricemia.

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ABSTRACT

Continuous development of medicine has so far failed to reduce the number of patients suffering from hypertension and hyperuricemia simultaneously. These patients are very threatened by the development of thrombosis of different localization in them. The main reason for this is the development of hyperaggregation of blood cells in them, the nature of which has so far been poorly investigated. The aim is to assess the state of aggregation properties of platelets in patients with hypertension with hyperuricemia. We examined 41 patients of the second adult age (mean age 54,4 ± 2,4 years) with arterial hypertension of 1-2 degrees, risk 4 with hyperuricemia. The control group consisted of 26 clinically healthy people of the same age. All persons under supervision were given written informed consent to participate in the study. Biochemical, hematological and statistical methods of investigation were used in the work. A large frequency of thrombosis of various localizations is characteristic for this patient population and is closely related to the development of platelet hyperaggregation. At the heart of this disorder in conditions of a combination of arterial hypertension with hyperuricemia is the weakening of antioxidant protection of the plasma with the activation of lipid peroxidation processes in it. At the same time for individuals with hypertension and hyperuricemia, the attenuation of platelet disaggregation was characteristic. As a result, patients receive a sharply increased risk of thrombosis of any location, which can lead to disability and death.

Keywords: platelets, arterial hypertension, hyperuricemia, aggregation.

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ISSN: 0975-8585

INTRODUCTION

Despite all the efforts of medical science and practice, the wide prevalence among the population of the mature age of developed countries the combination of arterial hypertension (AH) and hyperuricemia does not tend to decrease [1,2]. This combination is very dangerous high frequency of development of fatal vascular thrombosis with it [3]. Because of the high frequency of these events, vasopathy always stands in these patients, the prevalence of which also does not decrease [4]. Vasopathy is manifested primarily by the weakening of vascular control over the aggregation of blood elements, which is an important cause of increased hemostatic processes leading to thrombosis [5,6,7]. Vasopathy is always manifested by the weakening of synthesis in the walls of the vessels of the disaggregants, primarily prostacyclin and nitric oxide [8,9]. Given the prevalence of hypertension with hyperuricemia, it seemed important from a scientific and practical point of view to assess the state of vascular control of platelet aggregation in this patient population [10].

The goal is to assess the state of aggregation properties of platelets in hypertensive 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 41 patients of the second mature age (mean age 54.4±2.4 years) with AH of the 1^{st}-2^{nd} degree [11] 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. 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.

The state of platelet aggregation (AT) was evaluated by the micro-method [15,16] in plasma obtained without venous occlusion in response to ADP ($0.5x10^{-4}$ M), collagen (dilution 1:2 of the basic 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 the platelet-rich plasma as standardized for the platelet count of $200×10^9$ platelets. Aggregational properties of platelets in intravascular conditions were determined using a phase contrast microscope. Considered 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 2.3 times, TBA-active products – in 1.5 times, being accompanied by suppression of antioxidant plasma activity in 1.5 times (Table).

ISSN: 0975-8585

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).

Table. Registered indicators in the surveyed

Registrated parameters	**Patients, n=41, M±m**	**Control, n=26, M±m**
acylhydroperoxides plasma, D_{233} /1ml	3.27±0.08	1.42±0.09 p<0.01
TBA-compounds, mcmol/l	5.38±0.12	3.56±0.07 p<0,01
antioxidant activity plasma, %	22.2±0.17	32.9±0.12 p<0.01
biochemical parameters of platelets		
cholesterol of platelets, mkmol/10^9 platelets	1.08±0.005	0,67±0,005 p<0,01
common phospholipids of platelets, mkmol/10^9 platelets	0.33±0.008	0,49±0,004 p<0,01
acylhydroperoxides of platelets, D_{233}/10^9 platelets	3.42±0.09	2,20±0,04 p<0,01
malonic dialdehyde of platelets, nmol/10^9 platelets	1.35±0.11	0,68±0,02 p<0,01
catalase of platelets, ME/10^9 platelets	5100.0±23.85	9790,0±20,10 p<0,01
superoxidismutase of platelets, ME/10^9 platelets	1085.0±7.49	1650,0±3,00 p<0,01
aggregation of platelets		
aggregation with ADP, s	25.2±0.19	41,0±0,12 p<0,01
aggregation with collagen, s	23.3±0.15	33,2±0,10 p<0,01
aggregation with thrombin, s	36.5±0.14	55,3±0,05 p<0,01
aggregation with ristomycin, s	28.0±0.16	45,2±0,06 p<0,01
aggregation with epinephrine, s	69.8±0.22	93,0±0,07 p<0,01
aggregation with ADP and epinephrine, s	21.2±0.17	34,5±0,04 p<0,01
aggregation with ADP and collagen, s	17.3±0.19	26,6±0,05 p<0,01
aggregation with epinephrine and collagen, s	13.2±0.17	29,2±0,12 p<0,01
The number of platelets in the aggregates, %	12.5±0.18	6,5±0,07 p<0,01
Number of little aggregates (in 100 free thrombocytes)	17.9±0.19	3,1±0,03 p<0,01
Number of medium and large aggregates (in 100 free thrombocytes)	1.65±0.08	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 hyperuricemia hypertension, 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 those with hypertension and hyperuricemia exceeded those of the control group.

Important significance in the development of rheological disturbances and thrombophilia in persons with AH and hyperuricemia belongs to aggregation increase of regular blood elements and especially – platelets [19,20]. At combination of AH and 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.

Amplification of AP is largely due to the weakening of their disaggregation properties [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 a consequence of a pronounced disruption of aggregation and disaggregation mechanisms of platelets [31,32].

CONCLUSION

Aggregation of platelets is an important component of maintaining homeostasis in the body. With various pathological processes, it can be disturbed, which is manifested by its amplification. These disorders are very common in cardiac pathology, including arterial hypertension. Its frequent combination with hyperuricemia prompted the author to evaluate the aggregation capacity of platelets in this contingent of patients. In the work it was revealed that for the combination of arterial hypertension with hyperuricemia characterized by a pronounced increase in the aggregation properties of platelets. This situation seriously violates the hemostatic balance in the body of such patients and forms at them the danger of thrombosis of any localization.

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