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Disorders Of Disaggregation Control Of Blood Vessels Over Platelets In Hypertensive Patients With Arterial Hypertension.

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ABSTRACT

Despite the success of medicine and extensive preventive examinations of the population, the number of patients suffering from arterial hypertension and having hyperuricemia remains quite a lot. These patients are very threatened by the development of thrombosis of different localization in them. The main reason for this is the presence of vasopathy in them, the nature of which has so far been poorly investigated. The goal is to assess the state of disaggregation effects of blood vessels on platelets in patients with hypertension with hyperuricemia. We examined 41 patients of the second mature age (mean age 54.4±2.4 years) with arterial hypertension of the 1st-2nd degree, risk 4 with 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 arterial hypertension with 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 arterial hypertension combination with hyperuricemia. The persons with arterial hypertension and 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, arterial hypertension, hyperuricemia, vascular wall, antiaggregation.

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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 [5-7]. 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 disaggregation effects of blood vessels on platelets in hypertensive 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 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 malondialdehyde (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 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 $p<0.05$.

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).

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 patients with hyperuricemia hypertension, an earlier onset of AT with inductors and their combinations was revealed (Table). Previously, AT was attacked with collagen, a little later with ADP, even later with ristomycin, thrombin and adrenaline. The onset of AT 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.

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All the patients were noted to have the decrease of vessels' disaggregative impacts on platelets (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
antioxidantactivityplasma, %	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
malonicdialdehyde 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 in intact plasma		
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 freethrombocytes)	1.65±0.08	0.14±0.03 p<0.01

cardiovascular control of platelet aggregation		
IAAVWwith ADP	1.22±0.14	1.53±0.16 p<0.01
IAAVWwith collagen	1.17±0.23	1.48±0.16 p<0.01
IAAVWwith thrombin, s	1.18±0.18	1.44±0.13 p<0.01
IAAVWwith ristomycin, s	1.24±0.05	1.56±0.11 p<0.01
IAAVWwith epinephrine	1.33±0.15	1.62±0.13 p<0.01
IAAVWwith ADP and epinephrine	1.27±0.19	1.49±0.12 p<0.01
IAAVWwith ADP and collagen	1.24±0.23	1.51±0.10 p<0.01
IAAVWwithepinephrine and collagen	1.19±0.16	1.53±0.11 p<0.01
The number of platelets in the aggregatesafter temporary venous occlusion, %	9.8±0.05	4.5±0.15 p<0.01
Number of little aggregates (in 100 free thrombocytes)after temporary venous occlusion	7.6±0.17	2.1±0.15 p<0.01
Number of medium and large aggregates (in 100 freethrombocytes)after temporary venous occlusion	0.23±0.008	0.02±0.005 p<0.01

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

In patients with hypertension and hyperuricemia, a decrease in IAAWW was found with individual inducers (for adrenaline 1,33±0,15, for ADP 1,22±0,14, for ristomycin 1,24±0,05, for collagen and thrombin 1,17±0,23 and 1,18±0,18, respectively) and with their combinations (for ADP and adrenaline 1,27±0,19, for ADP and collagen - 1,24±0,23, for adrenaline and collagen - 1.19±0.16). 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 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.

Reduction IAAVW with inductors and their combinations is associated with a simultaneous increase in AT 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 Willebrandfactor [29,30]. The accelerated onset of AT 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].

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CONCLUSION

Synthesis of substances in the walls of blood vessels is an important component of maintaining homeostasis in the body. With various pathological processes, it can be disturbed, which is manifested, among other things, by depression of the disaggregation effects of blood vessels on platelets. These disorders are very common in cardiac pathology, including arterial hypertension. Its frequent combination with hyperuricemia prompted the author to evaluate the disaggregation effects on platelets in this contingent of patients. In the work it was revealed that the combination of arterial hypertension with hyperuricemia is characterized by a pronounced weakening of the disaggregation effects of blood vessels on platelets. This situation seriously violates the hemostatic balance in the body and creates the risk of thrombosis of any localization.

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