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Platelet Aggregation In Patients With Arterial Hypertension With Abdominal Obesity And Dyslipidemia.

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ABSTRACT

Unfortunately, among the population of many countries the number of suffering simultaneously arterial hypertension, abdominal obesity and dyslipidemia increases. It was revealed that they are characterized by a high incidence of thrombosis. This is due to the presence of their thrombocytopathy, the characteristics of which have not yet been fully investigated. The goal is to clarify the features of aggregation properties of platelets in patients with arterial hypertension with abdominal obesity and dyslipidemia. We examined 47 patients of the second adult age (mean age 53.4 ± 2.5 years) with grade 1 arterial hypertension, risk 4 with abdominal obesity and dyslipidemia. 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 in the work. The high frequency of thromboses of various localizations peculiar to this patient population is in many respects closely related to the development of platelet hyperaggregation in them. This violation occurs largely due to the weakening of the antioxidant protection of the plasma with the activation of the processes of lipid peroxidation in it. Also, in individuals with hypertension and abdominal obesity and dyslipidemia, weakened 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, arterial hypertension, abdominal obesity, dyslipidemia, aggregation.

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INTRODUCTION

In modern society, there is a high incidence among arterial hypertension (AH) population, combined with abdominal obesity and dyslipidemia [1,2]. This combination promotes the development of vascular thrombosis in persons of mature age leading to disability, and sometimes to death [3]. It is recognized that at the heart of a high incidence of thrombosis patients almost always are disorders in the blood cells [4]. At the same time, there is an increase in the aggregation of blood cells, which activates hemostasis and creates conditions favoring thrombosis [5,6,7]. Under these conditions depression of sensitivity of blood cells to vascular disaggregants develops, the main of which are prostacyclin and nitric oxide [8,9]. Given the high prevalence of hypertension with abdominal obesity and dyslipidemia, it seemed important to evaluate the features of vascular control of platelet aggregation in these patients [10].

The goal is to clarify the features of aggregation properties of platelets in patients with AH with abdominal obesity and dyslipidemia.

MATERIAL AND METHODS

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

We examined 47 patients of the second mature age (mean age 53.4 ± 2.5 years) with AH of the 1st-2nd degree [11] with abdominal obesity and dyslipidemia. 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 severity of platelet aggregation (AP) was assessed using a visual micromethode [15, 16] in plasma obtained without overlapping the cuff to a vessel using ADP (0.5×10^{-4} M), collagen (1: 2 dilution of the base suspension), thrombin (0.125 U/ml), ristomycin (0.8 mg/ml), adrenaline (5.0×10^{-6} M) and with combinations of ADP and epinephrine; ADP and collagen; adrenaline and collagen at the same plasma concentrations standardized for platelet count to 200×10^9 platelets/liter. Aggregational properties of platelets inside the vessels were determined using a phase contrast microscope, taking into account the number of small, medium and large aggregates and the degree of platelet involvement in them in plasma taken without the use of temporary venous occlusion [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 2.35 times, TBA-active products – in 1.35 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 hypertension with abdominal obesity and dyslipidemia, acceleration of AP with inductors and their combinations was found (Table). In the past, AP occurred in response to collagen, a little later on ADP, even later on ristomycin, thrombin and adrenaline. AP in response to a combination of inducers also developed accelerated. The number of circulating patients with platelet aggregates and the platelet count in them in patients with AH, abdominal obesity and dyslipidemia exceeded the level of the control group.

Table. Registered indicators in the surveyed

Registrated parameters	Patients, n=47, M±m	Control, n=26, M±m
acylhydroperoxides plasma, D ₂₃₃ /1ml	3.34±0.09	1.42±0.09 p<0.01
TBA-compounds, mcmol/l	5.47±0.12	3.56±0.07 p<0,01
antioxidant activity plasma, %	21.2±0.19	32.9±0.12 p<0.01
biochemical parameters of platelets		
cholesterol of platelets, mkmol/10 ⁹ platelets	1.14±0.005	0,67±0,005 p<0,01
common phospholipids of platelets, mkmol/10 ⁹ platelets	0.32±0.014	0,49±0,004 p<0,01
acylhydroperoxides of platelets, D ₂₃₃ /10 ⁹ platelets	3.59±0.12	2,20±0,04 p<0,01
malonic dialdehyde of platelets, nmol/10 ⁹ platelets	1.47±0.16	0,68±0,02 p<0,01
catalase of platelets, ME/10 ⁹ platelets	5000.0±23.60	9790,0±20,10 p<0,01
superoxidismutase of platelets, ME/10 ⁹ platelets	1100.0±9.24	1650,0±3,00 p<0,01
aggregation of platelets in intact plasma		
aggregation with ADP, s	23.5±0.14	41,0±0,12 p<0,01
aggregation with collagen, s	21.7±0.17	33,2±0,10 p<0,01
aggregation with thrombin, s	36.0±0.15	55,3±0,05 p<0,01
aggregation with ristomycin, s	27.0±0.13	45,2±0,06 p<0,01
aggregation with epinephrine, s	69.5±0.25	93,0±0,07 p<0,01
aggregation with ADP and epinephrine, s	20.2±0.20	34,5±0,04 p<0,01
aggregation with ADP and collagen, s	17.0±0.12	26,6±0,05 p<0,01
aggregation with epinephrine and collagen, s	12.0±0.15	29,2±0,12 p<0,01
The number of platelets in the aggregates, %	11.6±0.12	6,5±0,07 p<0,01
Number of little aggregates (in 100 free thrombocytes)	15.0±0.21	3,1±0,03 p<0,01
Number of medium and large aggregates (in 100 free thrombocytes)	1.65±0.06	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 AH and abdominal obesity and dyslipidemia belongs to aggregation increase of regular blood elements and especially – platelets [19,20]. At combination of AH and abdominal obesity and dyslipidemia 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].

The growth of platelet aggregation is also largely due to the weakening of their ability to disaggregate [25,26]. Apparently, a serious cause of this can be the activation of LPO in plasma [27,28]. Previously, the development of AP with ristomycin in patients should be associated with increased synthesis in the walls of their vascular Willebrand factor [29,30]. The accelerated onset of AP on combinations of inductors and an excessive number of platelet aggregates in the blood in patients before and after venous occlusion is a consequence of the resulting weakening of the ability to aggregate platelets in vivo [31, 32].

CONCLUSION

The activity of aggregation of blood cells is an important component of maintaining homeostasis. In the case of the development of pathology, there is an inevitable increase in the aggregation capacity of thrombocytes. This is very often found in cardiac pathology and especially often with arterial hypertension. The high frequency of the combination of arterial hypertension with abdominal obesity dictated the need to evaluate the aggregation capacity of platelets in this contingent of patients. It was found out that in case of presence of arterial hypertension, abdominal obesity and dyslipidemia, the weakening of aggregation capacities of their thrombocytes is noted in patients. The revealed disorders are considered as a serious cause of activation in patients of hemostasis mechanisms and formation of thrombosis risk.

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