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Severity of disaggregation control of blood vessels over platelets in patients with arterial hypertension with abdominal obesity.

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ABSTRACT

In recent years, the number of patients with arterial hypertension and abdominal obesity has increased in the society. It becomes clear that the high incidence of thrombosis in this contingent of patients is largely due to the presence of vasopathy, the characteristics of which have so far been poorly investigated. The goal is to clarify the features of the disaggregation properties of blood vessels in relation to platelets in patients with arterial hypertension with abdominal obesity. We examined 55 patients of the second mature age (mean age 51.2±2.7 years) with arterial hypertension of the 1st-2nd degree, risk 4 with abdominal obesity. 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 abdominal obesity 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 abdominal obesity. The persons with arterial hypertension and abdominal obesity 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, abdominal obesity, vascular wall, antiaggregation.

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INTRODUCTION

In modern civilized society, the high frequency of arterial hypertension (AH), which is increasingly associated with abdominal obesity [1,2], is noted. This fact ensures a high incidence of development of vascular thrombosis in persons of mature age leading to widespread disability and mortality [3]. It becomes clear that the cause of a high incidence of thrombosis of any location in any contingent of patients is almost always vasopathy, the prevalence of which is now increasing [4]. As a result of the presence of vasopathy, there is a weakening of vascular control over the aggregation of blood elements, which stimulates the mechanisms of hemostasis and leads to the development of thrombosis [5,6,7]. In the course of the development of vasopathy, the synthesis in the vessels of the disaggregants weakens, the most important of which are prostacyclin and nitric oxide [8,9]. In view of the prevalence of hypertension with abdominal obesity, it seemed important from a scientific and practical point of view to study the state of vascular control over platelet aggregation in this patient population [10].

The goal is to clarify the features of the disaggregation properties of blood vessels in relation to platelets in patients with AH and abdominal obesity.

MATERIALS AND METHODS

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

We examined 55 patients of the second mature age (mean age 51.2±2.7 years) with AH of the 1^{st}-2^{nd} degree [11] with abdominal obesity. 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 activity of platelet aggregation (AP) was elucidated by visual micromethod [16] in plasma obtained without and with venous occlusion using ADP (0.5x10-4 M), collagen (1: 2 dilution of the base suspension), thrombin (0.125ed/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 concentrations in a platelet-rich plasma with a standardized platelet count of 200×10^9 tp. The value of the index of antiaggregatory activity of the vascular wall (IAASC) was calculated by dividing the time of development of AT in the plasma after venous occlusion for a time in intact plasma. The disaggregation effects of the vessel wall on the intravascular aggregation of platelets were determined using a phase contrast microscope in terms of the number of small, medium and large aggregates and the involvement of platelets in them in plasma taken without temporal venous occlusion and in plasma obtained on its background [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 2.2 times, TBA-active products – in 1.4 times, being accompanied by suppression of antioxidant plasma activity in 1.4 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 persons with hypertension with abdominal obesity, acceleration of development of AP with inductors and their combinations was revealed (Table). Most quickly, AP came with collagen, a little later with ADP, even later with ristomycin, thrombin and adrenaline. The development of AP with combinations of inductors was also accelerated. The number of free platelet aggregates in the blood and the level of platelet involvement in those with hypertension and abdominal obesity exceeded control figures.

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=55, M±m	Control, n=26, M±m
acylhydroperoxides plasma, D_{233} /1ml	3.19±0.07	1.42±0.09 p<0.01
TBA-compounds, umol/l	5.21±0.11	3.56±0.07 p<0,01
antioxidantactivityplasma, %	22.5±0.15	32.9±0.12 p<0.01
biochemical parameters of platelets		
cholesterol of platelets, umol/10^9 platelets	1.06±0.008	0,67±0,005 p<0,01
common phospholipids of platelets, umol/10^9 platelets	0.34±0.006	0,49±0,004 p<0,01
acylhydroperoxides of platelets, D_{233}/10^9 platelets	3.36±0.07	2,20±0,04 p<0,01
malonicdialdehyde of platelets, nmol/10^9 platelets	1.34±0.09	0,68±0,02 p<0,01
catalase of platelets, ME/10^9 platelets	5105.0±19.74	9790,0±20,10 p<0,01
superoxidismutase of platelets, ME/10^9 platelets	1152.0±8.15	1650,0±3,00 p<0,01
aggregation of platelets in intact plasma		
aggregation with ADP, s	25.7±0.10	41,0±0,12 p<0,01
aggregation with collagen, s	23.6±0.16	33,2±0,10 p<0,01
aggregation with thrombin, s	37.2±0.11	55,3±0,05 p<0,01
aggregation with ristomycin, s	28.6±0.09	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	21.0±0.12	34,5±0,04

		p<0,01
aggregation with ADP and collagen, s	17.5±0.10	26,6±0,05 p<0,01
aggregation with epinephrine and collagen, s	13.5±0.13	29,2±0,12 p<0,01
The number of platelets in the aggregates, %	12.3±0.14	6,5±0,07 p<0,01
Number of little aggregates (in 100 free thrombocytes)	16.0±0.18	3,1±0,03 p<0,01
Number of medium and large aggregates (in 100 free thrombocytes)	1.51±0.05	0,14±0,03 p<0,01
cardiovascular control of platelet aggregation		
IAAVWwith ADP	1.24±0.10	1,53±0,16 p<0,01
IAAVWwith collagen	1.18±0.19	1,48±0,16 p<0,01
IAAVWwith thrombin	1.19±0.14	1,44±0,13 p<0,01
IAAVWwith ristomycin	1.25±0.09	1,56±0,11 p<0,01
IAAVWwith epinephrine	1.32±0.17	1,62±0,13 p<0,01
IAAVWwith ADP and epinephrine	1.26±0.16	1,49±0,12 p<0,01
IAAVWwith ADP and collagen	1.25±0.20	1,51±0,10 p<0,01
IAAVWwithepinephrine and collagen	1.18±0.15	1,53±0,11 p<0,01
The number of platelets in the aggregatesafter temporary venous occlusion, %	9.9±0.08	4,5±0,15 p<0,01
Number of little aggregates (in 100 free thrombocytes) after temporary venous occlusion	7.2±0.10	2,1±0,15 p<0,01
Number of medium and large aggregates (in 100 free thrombocytes)after temporary venous occlusion	0.19±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.

In patients with hypertension and abdominal obesity, a decrease in IAAVW with individual inducers was found (for adrenaline 1.32±0.17, for ADP 1.24±0.10, for ristomycin 1.25 ±0.09, for collagen and thrombin 1.18±0.19 and 1.19±0.14, respectively) and with their combinations (for ADP and adrenaline 1.26±0.16, for ADP and collagen – 1.25±0.20, for adrenaline and collagen - 1.18±0.15). In a plasma taken after a temporary venous occlusion, the number of platelet aggregates of various sizes and the high involvement of platelets in them decreased little in the blood of patients.

Important significance in the development of rheological disturbances and thrombophilia in persons with AH and abdominal obesity belongs to aggregation increase of regular blood elements and especially –

platelets [19,20]. At combination of AH and abdominal obesity 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 caused by a simultaneous increase in AT and a weakening of the disaggregation properties of the vessels [25,26]. Apparently, an important reason for this is the activation of plasma LPO [27,28]. Acceleration of AT with ristomycin in patients is caused by increased synthesis in the walls of the vessels of von Willebrand factor [29,30]. The rapid development of AT in response to combinations of inducers and a large number of platelet aggregates in patients before and after venous occlusion is the result of a pronounced weakening of the disaggregation capacity of the vessels [31, 32].

CONCLUSION

Vascular hemostasis is an important component of maintaining homeostasis in the body. One of the manifestations of its disorders is the weakening of the disaggregation properties of blood vessels in relation to platelets. This phenomenon is very common in various cardiac pathologies, including arterial hypertension. Often its combination with abdominal obesity dictated the need to assess the disaggregation effects on platelets in this contingent of patients. It was revealed that in arterial hypertension with abdominal obesity, there was a marked weakening of the disaggregation effects of blood vessels on platelets. These disorders in the examined patients were a serious basis for activation of hemocoagulation processes and development of thromboses of any localization in them.

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