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The Degree Of Weakening Of Disaggregation Control Of Blood Vessels Over Platelets In Abdominal Obesity And Dyslipidemia.

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ABSTRACT

Continuous improvement of the quality of medical care to the population can not contain the growth in the population of developed countries of the prevalence of a combination of abdominal obesity and dyslipidemia. It is noticed that for these patients the high frequency of thromboses is very characteristic. It is customary to associate with the presence of their vasopathy, many of the features of which have not yet been studied thoroughly. The aim is to assess the level of disaggregation properties of blood vessels in relation to platelets in patients with abdominal obesity and dyslipidemia. We examined 41 patients of the second mature age (mean age 49.2±1.8 years) 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. There were applied biochemical, hematological and statistical methods of investigation. High thromboses' frequency of various localizations at abdominal obesity and dyslipidemia 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 abdominal obesity and dyslipidemia. The persons with abdominal obesity and dyslipidemia 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, abdominal obesity, dyslipidemia vascular wall, antiaggregation.

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INTRODUCTION

Among the population of industrially developed countries there is a high incidence of a combination of abdominal obesity and dyslipidemia [1,2]. It is noticed that in the presence of this combination, vascular thromboses leading to disability, and sometimes to death, develop very often [3]. This phenomenon is almost always based on vasopathy, the frequency of which is also growing now [4]. Functionally, a very significant manifestation of vasopathy is the weakening of vascular control over the aggregation of blood elements. This inevitably leads to activation of hemostasis and creates conditions favoring thrombosis [5,6,7]. Depression of synthesis in the vessels of disaggregants in conditions of vasopathy leads to a decrease in the level in the blood of prostacyclin and nitric oxide [8,9], which largely implements vasopathy. Given the prevalence of abdominal obesity and dyslipidemia, it seemed important to assess the features of vascular control over platelet aggregation in patients with this pathological combination [10].

The goal was to assess the level of disaggregation properties of blood vessels in relation to platelets in patients with abdominal obesity and dyslipidemia.

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 49.2 ± 1.8 years) with abdominal obesity and dyslipidemia [11]. The control group was composed of 26 clinically healthy people of the same age. All the learned 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) in the form of Agat-Med and acylhydroperoxides (AHP) [13]. Antioxidant properties of the liquid part of the blood were determined according to the level of its antioxidant activity [14].

LPO activity in studied regular blood elements was determined according to the amount of malon dialdehyde (MDA) in the reaction of AHP in them [13]. In the studied and resuspended regular blood elements, we estimated the levels of cholesterol by the enzymatic colorimetric method with the help of a kit of the "Vital Diagnostikum" and CPL according to the content of phosphorus in them.

Evidence of vascular walling, control of platelets, aggregation was detected according to its weakening in the test with temporal venous occlusion [15].

The activity of platelet aggregation (AP) in the study was assessed with the help of a visual micromethode [16] in plasma obtained without application of the cuff to the vessel and after temporary venous occlusion using ADP ($0.5x10^{-4}$ M), collagen (1: 2 dilution of the basic suspension), thrombin (0.125 units / ml), ristomycin (0.8 mg / ml), epinephrine (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. The value of the index of antiaggregatory activity of the vascular wall (IAAVW) was calculated in the course of dividing the duration of AP in the plasma after a temporary venous occlusion during its onset in intact plasma. Disaggregation effects of blood vessels on intravascular platelet aggregation were determined using a phase contrast microscope, taking into account the number of small, medium and large aggregates and the degree of involvement of platelets in plasma taken without the use of temporary venous occlusion and in plasma obtained with its application [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 2.3 times, TBA-active products – in 1.5 times, being accompanied by suppression of antioxidant plasma activity in 1.5 times (Table).

Table: Registered indicators in the surveyed

Registrated parameters	Patients, n=41, M±m	Control, n=26, M±m
acylhydroperoxides plasma, D_{233}/1ml	3.29±0.08	1.42±0.09 p<0.01
TBA-compounds, µmol/l	5.38±0.09	3.56±0.07 p<0,01
antioxidant activity plasma, %	21.8±0.23	32.9±0.12 p<0.01
biochemical parameters of platelets		
cholesterol of platelets, mkmol/10^9 platelets	1.08±0.007	0.67±0.005 p<0.01
common phospholipids of platelets, mkmol/10^9 platelets	0.34±0.012	0.49±0.004 p<0.01
acylhydroperoxides of platelets, D_{233}/10^9 platelets	3.42±0.10	2.20±0.04 p<0.01
malonic dialdehyde of platelets, nmol/10^9 platelets	1.41±0.12	0.68±0.02 p<0.01
catalase of platelets, ME/10^9 platelets	5600.0±25.82	9790.0±20.10 p<0.01
superoxidismutase of platelets, ME/10^9 platelets	1200.0±10.17	1650.0±3.00 p<0.01
aggregation of platelets in intact plasma		
aggregation with ADP, s	25.2±0.12	41.0±0.12 p<0.01
aggregation with collagen, s	23.8±0.16	33.2±0.10 p<0.01
aggregation with thrombin, s	37.4±0.13	55.3±0.05 p<0.01
aggregation with ristomycin, s	28.5±0.14	45.2±0.06 p<0.01
aggregation with epinephrine, s	71.4±0.20	93.0±0.07 p<0.01
aggregation with ADP and epinephrine, s	21.8±0.25	34.5±0.04 p<0.01
aggregation with ADP and collagen, s	18.7±0.16	26.6±0.05 p<0.01
aggregation with epinephrine and collagen, s	13.5±0.18	29.2±0.12 p<0.01
The number of platelets in the aggregates, %	10.2±0.15	6.5±0.07 p<0.01
Number of little aggregates (in 100 free thrombocytes)	13.8±0.20	3.1±0.03 p<0.01
Number of medium and large aggregates (in 100 free	1.62±0.07	0.14±0.03 p<0.01

thrombocytes)		
cardiovascular control of platelet aggregation		
IAAVW with ADP	1.23±0.15	1.53±0.16 p<0.01
IAAVW with collagen	1.18±0.20	1.48±0.16 p<0.01
IAAVW with thrombin	1.19±0.19	1.44±0.13 p<0,01
IAAVW with ristomycin	1.22±0.16	1.56±0.11 p<0.01
IAAVW with epinephrine	1.31±0.14	1.62±0.13 p<0.01
IAAVW with ADP and epinephrine	1.25±0.18	1.49±0.12 p<0.01
IAAVW with ADP and collagen	1.23±0.22	1.51±0.10 p<0.01
IAAVW with epinephrine and collagen	1.18±0.18	1.53±0.11 p<0.01
The number of platelets in the aggregates after temporary venous occlusion, %	10.0±0.10	4.5±0.15 p<0.01
Number of little aggregates (in 100 free thrombocytes) after temporary venous occlusion	7.6±0.12	2.1±0.15 p<0.01
Number of medium and large aggregates (in 100 free thrombocytes) after temporary venous occlusion	0.25±0.009	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 the examined patients 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 degree of platelet involvement in those with abdominal obesity and dyslipidemia significantly exceeded the level of the control group.

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

The observed patients with abdominal obesity and dyslipidemia showed a decrease in IAAVW with respect to individual inducers (for adrenaline 1.31±0.14, for ADP 1.23±0.15, for ristomycin 1.22±0.16, for collagen and thrombin 1.18±0.20 and 1.19±0.20, respectively) and with their combinations (for ADP and adrenaline 1.25±0.18, for ADP and collagen – 1.23±0.22, for adrenaline and collagen – 1.18±0.18). In the blood of patients, obtained in conditions of temporary venous occlusion, the content of thrombocyte aggregates of any size and high degree of involvement of platelets in them decreased slightly.

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

especially – platelets [19,20]. At combination of 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. The level of disaggregating impacts from the side of vascular wall [23,24] lowers simultaneously with it in respect of platelets.

Decrease in IAAVW level with respect to applied inductors and their combinations is caused by simultaneous strengthening of AP processes and weakening of disaggregation effects of vessels [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]. Accelerated onset of AP in response to the appearance in the environment of a combination of inducers 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 disaggregation properties of the vascular endothelium [31, 32].

CONCLUSION

Antiaggregatory properties of blood vessels are an important element in ensuring the liquid state of the blood. With the development of vasopathy, it is unavoidable in patients that there is a weakening of the disaggregation capacity of the vessels, including in relation to platelets. Very often it occurs in metabolic disorders, especially with abdominal obesity and dyslipidemia. The high incidence of this pathology is the reason for assessing the disaggregation capacity of blood vessels in relation to platelets in this patient population. It was found out that in the case of patients with abdominal obesity and dyslipidemia, a pronounced weakening of the disaggregation capacity of the blood vessels with respect to platelets is recorded. These manifestations of vasopathy should be considered as a serious cause of activation in patients with hemostasis mechanisms and the formation of a high risk of thrombosis [33,34,35].

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