ISSN: 0975-8585

Research Journal of Pharmaceutical, Biological and Chemical Sciences

Degree Of Violation Of Disaggregation Control Of Blood Vessels Over Platelets In Patients With Arterial Hypertension With Abdominal Obesity And Dyslipidemia.

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ABSTRACT

The progress of medicine cannot yet restrain the growth in the population of the mature age of people suffering from hypertension, abdominal obesity and dyslipidemia. It was revealed that they are characterized by a high incidence of thrombosis. This is due to the presence of their vasopathy, the characteristics of which have not yet been fully 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 and dyslipidemia.We examined 47 patients of the second mature age (mean age 53.4±2.5 years) with arterial hypertension of the 1st-2nd degree, risk 4 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 arterial hypertension with 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 arterial hypertension combination with abdominal obesity and dyslipidemia. The persons with arterial hypertension and 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, arterial hypertension, abdominal obesity, dyslipidemia vascular wall, antiaggregation.

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ISSN: 0975-8585

INTRODUCTION

In the modern world, there is still a high frequency among the mature population of arterial hypertension (AH), 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 the basis for a high incidence of thrombosis in patients is almost always vasopathy, the frequency of which is now increasing [4]. With the development of vasopathy, there is a weakening of vascular control over the aggregation of blood elements, which activates hemostasis and creates conditions conducive to thrombosis [5,6,7]. In conditions of vasopathy, depression of synthesis in the vessels of 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 determine the features of the disaggregation control of blood vessels over platelets in patients with AH 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 47 patients of the second mature age (mean age 53.4±2.5 years) with AH of the 1^{st}-2^{nd} 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 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 severity of platelet aggregation (AT) was assessed using a visual micromethode [16] in plasma obtained without overlapping the cuff on 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 (IAASC) was calculated in the course of dividing the duration of AT in the plasma after a temporary venous occlusion during its onset in intact plasma. The disaggregation effects of the 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 platelet involvement in plasma taken without the use of temporary venous occlusion and 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 $p<0.05$.

ISSN: 0975-8585

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 AT with inductors and their combinations was found (Table). In the past, AT occurred in response to collagen, a little later on ADP, even later on ristomycin, thrombin and adrenaline. AT 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.

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=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
antioxidantactivityplasma, %	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
malonicdialdehyde 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 freethrombocytes)	1.65±0.06	0.14±0.03 p<0.01
cardiovascular control of platelet aggregation		
IAAVWwith ADP	1.22±0.16	1.53±0.16 p<0.01
IAAVWwith collagen	1.16±0.17	1.48±0.16 p<0.01
IAAVWwith thrombin, s	1.17±0.22	1.44±0.13 p<0.01
IAAVWwith ristomycin, s	1.21±0.14	1.56±0.11 p<0.01
IAAVWwith epinephrine	1.29±0.15	1.62±0.13 p<0.01
IAAVWwith ADP and epinephrine	1.24±0.19	1.49±0.12 p<0.01
IAAVWwith ADP and collagen	1.22±0.26	1.51±0.10 p<0.01
IAAVWwithepinephrine and collagen	1.16±0.17	1.53±0.11 p<0.01
The number of platelets in the aggregatesafter temporary venous occlusion, %	10.5±0.09	4.5±0.15 p<0.01
Number of little aggregates (in 100 free thrombocytes)after temporary venous occlusion	7.9±0.17	2.1±0.15 p<0.01
Number of medium and large aggregates (in 100 freethrombocytes)after temporary venous occlusion	0.28±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.

Patients with AH, abdominal obesity and dyslipidemia showed a decrease in IAAWW with respect to individual inducers (for adrenaline 1.29±0.15, for ADP 1.22±0.16, for ristomycin1.21±0.14, for collagen and thrombin 1.16±0.17 and 1.17±0.22, respectively) and with their combinations (for ADP and adrenaline 1.24±0.19, for ADP and collagen - 1.22±0.26, for adrenaline and collagen - 1,16±0,17). 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 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. The level of disaggregating impacts from the side of vascular wall [23,24] lowers simultaneously with it in respect of platelets.

The decrease in IAAVW against inducers and their combinations is caused by simultaneous enhancement of the AT processes and a weakening of the disaggregation effects of the vessels [25,26]. Apparently, a serious cause of this can be the activation of LPO in plasma [27,28]. Previously, the development of AT with ristomycin in patients should be associated with increased synthesis in the walls of their vascular Willebrand factor [29,30]. The accelerated onset of AT on combinations of inducers and the excessive number of platelet aggregates in the blood in patients before and after venous occlusion is a consequence of the resulting weakening of the disaggregating properties of the vascular endothelium [31, 32].

CONCLUSION

Vascular control of aggregation in the blood is an important component of maintaining homeostasis. In the case of the development of vasopathy, the disaggregation capabilities of the vessels inevitably decrease, especially with respect to platelets. This is very often found in cardiac pathology and especially often with arterial hypertension. The high frequency of combination of arterial hypertension with abdominal obesity dictated the need to assess the disaggregation capacity of blood vessels in relation to platelets in this contingent of patients. It was found out that in the case of arterial hypertension, abdominal obesity and dyslipidemia, patients showed a marked weakening of disaggregation capacity in relation to platelets. 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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