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Aggregation Of Thrombocytes In Patients With Arterial Hypertension With Type 2 Diabetes Mellitus.

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ABSTRACT

In modern society, there is still a high prevalence of a combination of arterial hypertension and type 2 diabetes. The great danger of this combination of diseases is associated with the risk of developing against their background frequent thromboses, the mechanisms of which have not been studied sufficiently. The aim is to evaluate the aggregation capacity of platelets in arterial hypertension and type 2 diabetes mellitus. We examined 42 patients of the second adult age (mean age 49.3 ± 2.9 years) with arterial hypertension of 1-2 degrees, risk 4 with diabetes mellitus type 2. The control group consisted of 26 clinically healthy people of the same age. All examined persons gave written informed consent to participate in the study. Biochemical, hematological and statistical methods of investigation were used. The high incidence of thrombosis in various locations in hypertension in type 2 diabetes is closely related to the development of platelet hyperaggregation. At the heart of its development is the weakening of antioxidant protection of the plasma with activation of the processes of lipid peroxidation, which is very often the case when a combination of arterial hypertension with type 2 diabetes mellitus. It was found that people with arterial hypertension and type 2 diabetes mellitus have an obvious weakening of platelet disaggregation with an increase in their aggregation ability. 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, type 2 diabetes mellitus, aggregation.

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INTRODUCTION

Modern society pays great attention to the development of medical practice. At the same time, high prevalence of a combination of arterial hypertension (AH) with type 2 diabetes is still maintained in developed countries [1,2]. This contingent of patients is highly threatened by the development of vascular thrombosis, often leading to disability and death [3]. It is known that very often the cause of thrombosis in different patients is hyperaggregation, the occurrence of which in patients gradually increases [4]. It is known that this pathology causes an inevitable weakening of vascular control over the degree of aggregation of various blood elements and especially platelets, which causes the development of thromboses [5,6,7]. One of the manifestations of thrombocytopathy is the depression of the sensitivity of platelets to vascular disaggregants, the most active of which are prostacyclin and nitric oxide [8,9]. Given the high frequency of occurrence of hypertension with diabetes mellitus type 2, it seemed important for science and practice to assess the level of platelet aggregation in such patients [10].

The goal is to assess the aggregation capacity of platelets in hypertension with diabetes mellitus type 2.

MATERIAL AND METHODS

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

We examined 42 patients of the second mature age (mean age 49.3±2.9 years) with AH of the 1^{st}-2^{nd} degree [11] with type 2 diabetes mellitus. 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 common phospholipids (CPL) according to the content of phosphorus in them.

The severity of platelet aggregation (AT) was assessed using a visual micromethode [15] using ADP ($0.5x10^{-4}$ M), collagen (1: 2 dilution of the base suspension), thrombin (0.125 U/ml), ristomycin (0.8 mg/ml), epinephrine ($5.0×10^{-6}$ M) and with combinations of ADP and epinephrine; ADP and collagen; epinephrine and collagen in the same doses in a platelet-rich plasma, standardized by the number of platelets to $200×10^9$ platelets [16]. The level of platelet aggregation processes in intravascular conditions was determined using a phase contrast microscope and considering the number of small, medium and large aggregates and the degree of platelet involvement in them in plasma [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 1.54 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).

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In persons with AH with type 2 diabetes mellitus, acceleration of occurrence of AP with all inducers and their combinations was found (Table). The earliest time the AT developed with collagen, a little later with ADP, even later with ristomycin, thrombin and adrenaline. The onset of AP with the tested combinations of inductors was greatly accelerated. The value of free-flowing patients with platelet aggregates and the degree of involvement of platelets in hypertension with diabetes mellitus type 2 exceeded control figures.

Table. Registered indicators in the surveyed

Registrated parameters	Patients, n=42, 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±0.16	3.56±0.07 p<0,01
antioxidant activity plasma, %	21.3±0.18	32.9±0.12 p<0.01
biochemical parameters of platelets		
cholesterol of platelets, mkmol/10^9 platelets	1.11±0.005	0,67±0,005 p<0,01
common phospholipids of platelets, mkmol/10^9 platelets	0.32±0.011	0,49±0,004 p<0,01
acylhydroperoxides of platelets, D_{233}/10^9 platelets	3.48±0.09	2,20±0,04 p<0,01
malonic dialdehyde of platelets, nmol/10^9 platelets	1.48±0.10	0,68±0,02 p<0,01
catalase of platelets, ME/10^9 platelets	4910.0±24.95	9790,0±20,10 p<0,01
superoxidismutase of platelets, ME/10^9 platelets	1110.0±9.26	1650,0±3,00 p<0,01
aggregation of platelets		
aggregation with ADP, s	23.2±0.16	41,0±0,12 p<0,01
aggregation with collagen, s	21.4±0.17	33,2±0,10 p<0,01
aggregation with thrombin, s	35.0±0.15	55,3±0,05 p<0,01
aggregation with ristomycin, s	27.0±0.14	45,2±0,06 p<0,01
aggregation with epinephrine, s	70.1±0.12	93,0±0,07 p<0,01
aggregation with ADP and epinephrine, s	19.0±0.17	34,5±0,04 p<0,01
aggregation with ADP and collagen, s	15.2±0.16	26,6±0,05 p<0,01
aggregation with epinephrine and collagen, s	12.3±0.22	29,2±0,12 p<0,01
The number of platelets in the aggregates, %	15.0±0.19	6,5±0,07 p<0,01
Number of little aggregates (in 100 free thrombocytes)	18.2±0.21	3,1±0,03 p<0,01
Number of medium and large aggregates (in 100 free thrombocytes)	1.64±0.07	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 type 2 diabetes mellitus belongs to aggregation increase of regular blood elements and especially – platelets [19,20]. At combination of AH and type 2 diabetes mellitus 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. At the same time, with respect to platelets, the level of aggregating properties of platelets decreases [23,24].

Activation of platelet aggregation significantly worsened the rheology of blood in the capillaries [25,26]. An important reason for this is the activation of LPO in plasma [27,28]. The early onset of AT in response to ristomycin in patients is associated with an increase in sensitivity to the von Willebrand factor [29,30]. The accelerated onset of AT in response to two inducers and an excessive number of platelet aggregates in the blood of patients before and after temporary venous occlusion should be considered a manifestation of increasing depression of platelet disaggregation mechanisms [31, 32].

CONCLUSION

Platelet aggregation is very important for the optimal functioning of the body. A serious manifestation of its dysfunction is its amplification in all vessels. This can be noted especially often in patients with cardiac pathology, including arterial hypertension. Recently, it is often combined with type 2 diabetes mellitus, which is accompanied by a high incidence of thrombosis in this contingent of patients. It is established that in arterial hypertension with type 2 diabetes mellitus, there is often a strong activation of platelet aggregation functions. These disorders were a serious cause of activation of hemostasis mechanisms in patients with arterial hypertension and type 2 diabetes and development of the risk of fatal thrombosis.

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