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Anti-aggregation Effects Of Blood Vessels On Platelets In Patients With Dyslipidemia With Impaired Glucose Tolerance.

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ABSTRACT

Many researchers now note the increase in the number of patients in the society suffering from both dyslipidemia and impaired glucose tolerance. They have a high incidence of vascular thrombosis, which is usually associated with the presence of vasopathy, the characteristics of which have not yet been adequately studied. The goal is to find out the level of disaggregation capacity of blood vessels in relation to platelets in patients with dyslipidemia with impaired glucose tolerance. We examined 45 patients of the second mature age (mean age 47.6±1.5 years) with dyslipidemia 4 with impaired glucose tolerance. 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 dyslipidemia with impaired glucose toleranceis 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 impaired glucose tolerance. The persons with dyslipidemia and impaired glucose tolerance 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, dyslipidemia, impaired glucose tolerance, vascular wall, antiaggregation.

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INTRODUCTION

It is known that at present in many countries there is a high prevalence of dyslipidemia, combined with a violation of glucose tolerance [1,2]. This combination is accompanied by a very high risk of development in these patients of various vascular thromboses, leading to frequent disability and mortality [3]. It is noted that the onset of thrombosis of any localization is always associated with the presence of vasopathy, which now occurs increasingly [4]. At the same time, vasopathy is always manifested, including the weakening of vascular control over the processes of aggregation of blood cells. This circumstance strongly stimulates various mechanisms of hemostasis, thereby leading to thrombosis [5,6,7]. In this connection, vasopathy manifests itself in a different degree of weakening of the synthesis of desaggregant substances in the vessels, the most important of which are prostacyclin and nitric oxide [8,9]. In view of the wide prevalence of the combination of dyslipidemia with impaired glucose tolerance, it was very important to evaluate the state of vascular control over platelet aggregation in these patients [10].

The goal is to find out the level of disaggregation capacity of blood vessels in relation to platelets in patients with dyslipidemia and impaired glucose tolerance.

MATERIALS AND METHODS

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

We examined 45 patients of the second mature age (mean age 47.6±1.5 years) with dyslipidemia with impaired glucose tolerance [11]. 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.

Evidence of vascular wall's control over platelets' aggregation was detected according to its weakening in the test with temporal venous occlusion [15].

The level of platelet aggregation (AP) was assessed by visual micromethod [16] in plasma obtained without and using 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), epinephrine (5.0×10^{-6} M) and with combinations of ADP and epinephrine; ADP and collagen; adrenaline and collagen at the same concentrations in the platelet-rich plasma as standardized for the platelet count of 200×10^9 platelets. The value of the index of antiaggregatory activity of the vascular wall (IAAVW) was calculated during the division of the time of development of AP in the plasma after venous occlusion during the time of this process in intact plasma. The severity of disaggregation capacity of blood vessels with respect to intravascular aggregation of platelets was determined using a phase contrast microscope by recording the number of small, medium and large aggregates and the degree of platelet involvement in them in plasma obtained without temporal venous occlusion and in plasma obtained against 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 2.1 times, TBA-active products – in 1.4 times, being accompanied by suppression of antioxidant plasma activity in 1.33 times (Table).

Table. Registered indicators in the surveyed

Registrated parameters	Patients, n=45, M±m	Control, n=26, M±m
acylhydroperoxides plasma, D_{233}/1ml	3.02±0.09	1.42±0.09 p<0.01
TBA-compounds, umol/l	4.99±0.16	3.56±0.07 p<0,01
antioxidant activity plasma, %	24.8±0.22	32.9±0.12 p<0.01
biochemical parameters of platelets		
cholesterol of platelets, umol/10^9 platelets	1.09±0.014	0.67±0.005 p<0.01
common phospholipids of platelets, umol/10^9 platelets	0.34±0.008	0.49±0.004 p<0.01
acylhydroperoxides of platelets, D_{233}/10^9 platelets	3.51±0.05	2.20±0.04 p<0.01
malonic dialdehyde of platelets, nmol/10^9 platelets	1.39±0.13	0.68±0.02 p<0.01
catalase of platelets, ME/10^9 platelets	5200.0±26.11	9790.0±20.10 p<0.01
superoxidismutase of platelets, ME/10^9 platelets	1100.0±8.36	1650.0±3.00 p<0.01
aggregation of platelets in intact plasma		
aggregation with ADP, s	25.7±0.14	41.0±0.12 p<0.01
aggregation with collagen, s	23.4±0.12	33.2±0.10 p<0.01
aggregation with thrombin, s	36.2±0.13	55.3±0.05 p<0.01
aggregation with ristomycin, s	27.4±0.05	45.2±0.06 p<0.01
aggregation with epinephrine, s	71.5±0.12	93.0±0.07 p<0.01
aggregation with ADP and epinephrine, s	20.3±0.16	34.5±0.04 p<0.01
aggregation with ADP and collagen, s	16.5±0.07	26.6±0.05 p<0.01
aggregation with epinephrine and collagen, s	13.0±0.12	29.2±0.12 p<0.01
The number of platelets in the aggregates, %	13.1±0.15	6.5±0.07 p<0.01
Number of little aggregates (in 100 free thrombocytes)	17.4±0.18	3.1±0.03 p<0.01
Number of medium and large aggregates (in 100 free thrombocytes)	1.54±0.09	0.14±0.03 p<0.01
vascular control of platelet aggregation		
IAAVW with ADP	1.26±0.14	1.53±0.16 p<0.01
IAAVW with collagen	1.20±0.18	1.48±0.16 p<0.01

IAAVW with thrombin	1.17±0.24	1.44±0.13 p<0.01
IAAVW with ristomycin	1.25±0.16	1.56±0.11 p<0.01
IAAVW with epinephrine	1.32±0.22	1.62±0.13 p<0.01
IAAVW with ADP and epinephrine	1.28±0.19	1.49±0.12 p<0.01
IAAVW with ADP and collagen	1.27±0.22	1.51±0.10 p<0,01
IAAVW with epinephrine and collagen	1.19±0.15	1.53±0.11 p<0.01
The number of platelets in the aggregates after temporary venous occlusion, %	11.0±0.12	4.5±0.15 p<0.01
Number of little aggregates (in 100 free thrombocytes) after temporary venous occlusion	7.5±0.21	2.1±0.15 p<0.01
Number of medium and large aggregates (in 100 free thrombocytes) after temporary venous occlusion	0.20±0.007	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 patients with dyslipidemia and impaired glucose tolerance, the development of AP with inductors and their combinations was revealed (Table). The earliest time 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 further accelerated. The number of platelet aggregates and the level of platelet involvement in patients with dyslipidemia and impaired glucose tolerance exceeded the control figures.

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

In patients with dyslipidemia and impaired glucose tolerance, a decrease in IAAVW with individual inducers was found (for adrenaline 1.32±0.22, for ADP 1.26±0.14, for ristomycin 1.25±0.16, for collagen and thrombin 1.20±0.18 and 1.17±0.24, respectively) and with their combinations (for ADP and adrenaline 1.28±0.19, for ADP and collagen – 1.27±0.22, for adrenaline and collagen – 1.19±0.15). In the blood of patients, taken after a temporary venous occlusion, the number of platelet aggregates of various sizes and the degree of involvement of platelets in them decreased slightly.

Important significance in the development of rheological disturbances and thrombophilia in persons with dyslipidemia and impaired glucose tolerance belongs to aggregation increase of regular blood elements and especially – platelets [19,20]. At combination of dyslipidemia and impaired glucose tolerance 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 with inductors and their combinations is caused not only by the increase in AP, but also by the weakening of the disaggregation capacity of the vessels [25,26]. Apparently, an important role in this is the activation of LPO in plasma [27,28]. Acceleration of the process of AP with ristomycin in patients is associated with increased synthesis in the walls of the vessels of von Willebrand factor and growth of its content in their plasma [29,30]. A rapid onset in patients with AP in response to combinations of two

inducers and the presence of a large number of platelet aggregates in their blood before and after venous occlusion is a consequence of severe depression of the disaggregation mechanisms of their vessels [31, 32].

CONCLUSION

The disaggregation properties of the vessels are extremely important for maintaining homeostasis in the body. A dangerous manifestation of his disorders is the weakening of the antiaggregational capacity of blood vessels in relation to platelets. These disorders are very common in any metabolic pathology, including dyslipidemia and impaired glucose tolerance. This dictates the need to clarify the disaggregation capacity of blood vessels in relation to platelets in this contingent of patients. It was found that with dyslipidemia with a violation of glucose tolerance there is a pronounced weakening of the antiaggregational effects of blood vessels on platelets. These disorders in this contingent of patients are a serious cause of activation of hemostasis in them and development of a risk of thrombosis of any localization [33,34,35].

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