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Disaggregation Properties Of Blood Vessels In Relation To Platelets In Patients With Dyslipidemia.

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ABSTRACT

Long-term observations have shown that among the population of industrially developed countries, the prevalence of dyslipidemia persists. At the heart of the high frequency of vascular thrombosis in this category of patients often are the phenomena of vasopathy, the severity of which has recently been gradually calculated. The goal is to evaluate the disaggregation capacity of blood vessels in relation to platelets in patients with dyslipidemia. We examined 380 patients of the second mature age (mean age 53.8±1.6 years) with dislipidemia of IIb type. 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 dislipidemia 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 dislipidemia. The persons with dislipidemia 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, dislipidemia, vascular wall, antiaggregation.

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INTRODUCTION

The rise in the standard of living of the population of industrially developed countries leads to a widespread prevalence of nutrition in them with an excessive amount of fat leading to dyslipidemia [1,2]. This leads to an increase in the incidence of vascular thrombosis in persons of working age leading to disability and often to death [3]. At the heart of this in this contingent of patients, as a rule, is vasopathy, the severity of which tends to increase [4]. An important manifestation of vasopathy is the weakening of vascular control over the aggregation of blood cells, which strongly stimulates the processes of hemostasis and leads to the development of thrombosis [5,6,7]. This is due to the weakening of synthesis in the wall of the vessels of the disaggregants [8,9]. Considering the wide prevalence of dyslipidemia, it is of great interest to study the level of vascular control over platelet aggregation in this contingent of patients [10]. The aim of the study is to assess the disaggregation capacity of blood vessels in relation to platelets in patients with dyslipidemia.

MATERIALS AND METHODS

The research was approved by the Ethics Committee of Kursk Institute of Social Education (branch of RussianStateSocialUniversity) (record №5 from 12.05.2014).

We examined 41 patients of the second mature age (mean age 53.8±1.6 years) with dislipidemia of IIb type [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.

We determined the content of common cholesterol (CS) and triglycerides (TG) in blood of all the observed persons by enzymatic colorimetric method with the help of a kit “Vital Diagnostikum” (Russia). CS level of high-density lipoproteins (HDLP) was determined with the help of a kit “OlveksDiagnostikum (Russia) by enzymatic colorimetric method. Common lipids (CL) were estimated with the help of a kit “Erba Russ” (Russia). The quantity of common phospholipids (CPL) in blood plasma was registered according to the content of phosphorus in them. CS levels of low-density lipoproteins (LDLP) were established by calculation according to Freedwald V. CS concentrations of very low-density lipoproteins (VLDLP) was determined according to the formula: TG content/2.2. Received indices of common CS and CS of LDLP were considered as normal, border-line or high in accordance with Russian recommendations (2012) [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 (AOA) [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 level of platelet aggregation (AP) was assessed by visual micromethod [16] before and after 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 index of antiaggregatory activity of the vascular wall (IAASC) was calculated by dividing the time of development of AP after venous occlusion for a time without it. Disaggregation capacity of the vessel wall over intravascular aggregation of platelets was determined using a phase contrast microscope in terms of the number of small, medium and large aggregates and the involvement of platelets in them before and after temporary venous occlusion [17,18].

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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 $p<0.05$.

RESULTS AND DISCUSSION

The blood of patients was noted to have levels' increase of CL and common CS which surpassed the control values in 1.6 and 1.3 times, respectively, at simultaneous lowering of plasma CPL in 2.1 times (Table). The blood of persons with dislipidemia was found to have the increase of CS LDLP, CS VLDLP and TG in 1.64, 1.61 and 1.60 times, respectively. It is combined with the lowering of CS HDLP in 1.55 times. 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 1.3 times (Table).

Table: Registered indicators in the surveyed

Registrated parameters	Patients with arterial hypertension and dyslipidemia, n=41, M±m	Control, n=26, M±m
common cholesterol, mmol/l	6.3±0.05	4.8±0.05 p<0.01
CS level of high-density lipoproteins, mmol/l	1.07±0.06	1.60±0.06 p<0.01
CS levels of low-density lipoproteins, mmol/l	3.99±0.07	2.43±0.04 p<0.01
CS concentrations of very low-density lipoproteins, mmol/l	1.24±0.04	0.77±0.05 p<0.01
triglycerides, mmol/l	2.72±0.06	1.70±0.02 p<0.01
common lipids, g/l	9.0±0.10	5.6±0.03 p<0.01
common phospholipids, mmol/l	1.70±0.06	3.54±0.09 p<0.01
acylhydroperoxides plasma, D_{233} /1ml	3.01±0.07	1.42±0.09 p<0.01
TBA-compounds, mcmol/l	4.92±0.08	3.56±0.07 p<0,01
antioxidantactivityplasma, %	24.2±0.13	32.9±0.12 p<0.01
biochemical parameters of platelets		
cholesterol of platelets, mkmol/10^9 platelets	1.02±0.006	0.67±0.005 p<0.01
common phospholipids of platelets, mkmol/10^9 platelets	0.34±0.003	0.49±0.004 p<0.01
acylhydroperoxides of platelets, D_{233}/10^9 platelets	3.06±0.04	2.20±0.04 p<0.01
malonicdialdehyde of platelets, nmol/10^9 platelets	1.28±0.07	0.68±0.02 p<0.01
catalase of platelets, ME/10^9 platelets	5350.0±17.05	9790.0±20.10 p<0.01
superoxidismutase of platelets, ME/10^9 platelets	1200.0±6.30	1650.0±3.00 p<0.01

aggregation of platelets in intact plasma		
aggregation with ADP, s	25.3±0.08	41.0±0.12 p<0.01
aggregation with collagen, s	23.4±0.10	33.2±0.10 p<0.01
aggregation with thrombin, s	36.7±0.13	55.3±0.05 p<0.01
aggregation with ristomycin, s	28.7±0.10	45.2±0.06 p<0.01
aggregation with epinephrine, s	72.4±0.14	93.0±0.07 p<0.01
aggregation with ADP and epinephrine, s	21.3±0.16	34.5±0.04 p<0.01
aggregation with ADP and collagen, s	18.5±0.12	26.6±0.05 p<0.01
aggregation with epinephrine and collagen, s	16.2±0.11	29.2±0.12 p<0.01
The number of platelets in the aggregates, %	10.0±0.14	6.5±0.07 p<0.01
Number of little aggregates (in 100 free thrombocytes)	11.2±0.10	3.1±0.03 p<0.01
Number of medium and large aggregates (in 100 free thrombocytes)	1.28±0.09	0.14±0.03 p<0.01
cardiovascular control of platelet aggregation		
IAAVWwithADP	1.27±0.10	1.53±0.16 p<0.01
IAAVWwith collagen	1.20±0.12	1.48±0.16 p<0.01
IAAVWwith thrombin, s	1.21±0.14	1.44±0.13 p<0.01
IAAVWwith ristomycin, s	1.26±0.13	1.56±0.11 p<0.01
IAAVWwithepinephrine	1.38±0.16	1.62±0.13 p<0.01
IAAVWwith ADP and epinephrine	1.27±0.10	1.49±0.12 p<0.01
IAAVWwith ADP and collagen	1.26±0.09	1.51±0.10 p<0.01
IAAVWwithepinephrine and collagen	1.19±0.11	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	6.9±0.09	2.1±0.15 p<0.01
Number of medium and large aggregates (in 100 free thrombocytes)after temporary venous occlusion	0.17±0.004	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 patients with dyslipidemia, acceleration of development of AP with individual inducers and their combinations was found (Table). The earliest time the AP came under the action of collagen, a little later with ADP, even later with ristomycin, thrombin and adrenaline. The AP in response to a combination of inductors was also accelerated. The number of free-circulating patients with platelet aggregates of different sizes and the degree of platelet involvement in them in persons with dyslipidemia significantly exceeded the control values.

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

In individuals with dyslipidemia, a decrease in IAAID with a single agonist was noted (for adrenaline 1.38±0.16, for ADP 1.27±0.10, for ristomycin 1.26±0.13, for collagen and thrombin 1.20±0.12 and 1.21±0.14, respectively) and with their combinations (for ADP and adrenaline 1.27±0.10, for ADP and collagen – 1.26±0.09, for adrenaline and collagen – 1.19± 0.11). This was accompanied by an increase in the amount of thrombocyte aggregates of various sizes and excessive involvement of platelets against the background of temporary venous occlusion.

Important significance in the development of rheological disturbances and thrombophilia in persons with dislipidemia belongs to aggregation increase of regular blood elements and especially – platelets [19,20]. At dislipidemia 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 of the values of IAASS with individual inducers and their combinations is based on the simultaneous enhancement of AT and the weakening of disaggregation effects on them from the side of the vascular wall [25,26]. Apparently, this is caused by dyslipidemia and activation of LPO in plasma [27,28]. Acceleration of AT in response to ristomycin in patients is associated with increased synthesis in the vascular endothelium of von Willebrand factor [29,30]. Acceleration of AT in response to combinations of inducers and an excessive number of platelet aggregates in the blood of patients before and after venous occlusion indicated a marked weakening of the disaggregation effects of blood vessels on platelets [31, 32].

CONCLUSION

High thromboses' frequency of various localizations with dislipidemia is closely connected with angiopathy development against their background. Weakening of plasma antioxidant protection with activation of LPO processes in it leading to alteration of vascular wall, is noted in conditions of dislipidemia. The persons with dislipidemia 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 [33,34,35].

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