ISSN: 0975-8585

Research Journal of Pharmaceutical, Biological and Chemical Sciences

Aggregational Activity Of Thrombocytes In Patients With Type 2 Diabetes Mellitus.

Medvedev IN*.

Russian State Social University, st. V. Pika, 4, Moscow, Russia, 129226.

ABSTRACT

High pathological burden of modern society is largely associated with the prevalence among the general population of the world of type 2 diabetes. The special danger of this disease is associated with the frequent development of thromboses caused by hyperaggregation of blood cells, the nature of which has not been studied sufficiently. The goal is to assess the level of platelet aggregation in type 2 diabetes mellitus. We examined 36 patients of the second adulthood (mean age 47.4 ± 2.1 years) with type 2 diabetes mellitus. 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 frequency of high thrombosis of various localizations in type 2 diabetes is closely related to the development of angiopathy against their background. Weakening of antioxidant protection of plasma with activation of processes of lipid peroxidation in it, leading to a change in the vascular wall, is noted in conditions of type 2 diabetes mellitus. It was found that people with type 2 diabetes mellitus have an obvious weakening of disaggregation of vascular wall vascular impacts with increasing platelet aggregation capacity. As a result, patients receive a sharply increased risk of thrombosis of any location, which can lead to disability and death.

Keywords: platelets, thrombocytopathy, type 2 diabetes mellitus, pathology, aggregation.

Corresponding author

ISSN: 0975-8585

INTRODUCTION

At present, violations of carbohydrate metabolism in the form of type 2 diabetes mellitus still retain a high prevalence [1,2]. Earlier it was noted that in this contingent of patients vascular thromboses often lead to disability and early death [3]. Previous studies have also shown that the basis of these thromboses often lies in the hyperaggregation of blood cells, the occurrence of which in these patients has recently increased [4]. It is known that hyperaggregation of blood cells is manifested by a weakening of their sensitivity to disaggregants and activation of hemostasis mechanisms [5,6,7]. They are based on violations of blood cell receptors, primarily to prostacyclin and nitrogen oxide [8,9]. Given the high incidence of type 2 diabetes mellitus, it seemed important to assess the level of platelet aggregation in such patients [10].

In this regard, in this paper, the goal is to evaluate the level of platelet aggregation in type 2 diabetes mellitus.

MATERIAL AND METHODS

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

We examined 36 patients of the second mature age (mean age 47.4±2.1 years) with type 2 diabetes mellitus [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 total phospholipids according to the content of phosphorus in them.

The activity of the platelet aggregation process (AP) was assessed using a visual micromethode [15] in plasma obtained after temporary venous occlusion and without it 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 intravascular aggregation of platelets was determined using a phase contrast microscope and taking into account 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 2.2 times, TBA-active products – in 1.5 times, being accompanied by suppression of antioxidant plasma activity in 1.4 times (Table).

The observed patients were noted to have increased cholesterol content in erythrocytes' membranes which was accompanied by the decrease of total phospholipids in them and LPO activation on behalf of weakening of their antioxidant protection (Table).

ISSN: 0975-8585

Table. Registered indicators in the surveyed

Registrated parameters	Patients, n=36, M±m	Control, n=26, M±m
acylhydroperoxides plasma, D_{233}/1ml	3.10±0.10	1.42±0.09 p<0.01
TBA-compounds, µmol/l	5.34±0.14	3.56±0.07 p<0,01
antioxidant activity plasma, %	23.8±0.16	32.9±0.12 p<0.01
biochemical parameters of platelets		
cholesterol of platelets, umol/10^9 platelets	1.02±0.002	0,67±0,005 p<0,01
common phospholipids of platelets, µmol/10^9 platelets	0.34±0.014	0,49±0,004 p<0,01
acylhydroperoxides of platelets, D_{233}/10^9 platelets	3.32±0.08	2,20±0,04 p<0,01
malonic dialdehyde of platelets, nmol/10^9 platelets	1.40±0.15	0,68±0,02 p<0,01
catalase of platelets, ME/10^9 platelets	5400.0±20.31	9790,0±20,10 p<0,01
superoxidismutase of platelets, ME/10^9 platelets	1250.0±8.62	1650,0±3,00 p<0,01
aggregation of platelets		
aggregation with ADP, s	25.7±0.15	41,0±0,12 p<0,01
aggregation with collagen, s	22.0±0.16	33,2±0,10 p<0,01
aggregation with thrombin, s	38.6±0.18	55,3±0,05 p<0,01
aggregation with ristomycin, s	29.1±0.12	45,2±0,06 p<0,01
aggregation with epinephrine, s	74.2±0.14	93,0±0,07 p<0,01
aggregation with ADP and epinephrine, s	21.0±0.13	34,5±0,04 p<0,01
aggregation with ADP and collagen, s	16.3±0.14	26,6±0,05 p<0,01
aggregation with epinephrine and collagen, s	14.1±0.18	29,2±0,12 p<0,01
The number of platelets in the aggregates, %	13.2±0.23	6,5±0,07 p<0,01
Number of little aggregates (in 100 free thrombocytes)	17.0±0.24	3,1±0,03 p<0,01
Number of medium and large aggregates (in 100 free thrombocytes)	1.51±0.05	0,14±0,03 p<0,01

Note: p - reliability of differences in the indices of a group of patients and a control group.

In patients with type 2 diabetes mellitus, the onset of AP with all inducers and their combinations was accelerated (Table). The earliest time the AP 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 importance of blood-free patients with platelet aggregates and the degree of platelet involvement in diabetes mellitus type 2 exceeded control figures.

Important implications in the development of rheological disturbances and thrombophilia in persons with type 2 diabetes mellitus belongs to the aggregation of regular blood elements and especially platelets [19,20]. At type 2 diabetes mellitus the depression of plasma antioxidant activity [21,22]. The effects of platelet membranes. The development of these manifestations in combination with these patients' platelets. Lipid imbalance leads to their hyperaggregability. At the same time, the ability to disaggregate in platelets is reduced [23,24].

Amplification of AP with inductors and their combinations is caused by the growth of AP mechanisms and depression of mechanisms of platelet disaggregation [25,26]. An important reason for this is the activation of LPO in plasma [27,28]. Acceleration of AT in response to ristomycin in patients is associated with increased factor of von Willebrand generation in the vessels [29,30]. Previously, the development of AP in response to two inducers and an excessive number of platelet aggregates in the blood of patients should be considered a manifestation of increasing depression of the disaggregation capacity of platelets [31, 32].

CONCLUSION

An important manifestation of type 2 diabetes is the hyperaggregation of blood cells. Recently, it often leads to various vascular thrombosis. The study found that in patients with type 2 diabetes mellitus, there is a weakening of the disaggregation properties of platelets. These disorders inevitably lead to the activation of hemostasis mechanisms in patients with type 2 diabetes and often contribute to the development of vascular complications [33,34,35].

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