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Expression Of Spontaneous Aggregation Of Erythrocytes In Patients With Arterial Hypertension With Hyperuricemia.

Medvedev IN*.

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

ABSTRACT

The very wide prevalence of thrombosis in hypertensive patients with hyperuricemia is largely associated with dysfunction of their blood cells. The situation is aggravated by the high frequency of occurrence in the developed countries of a combination of arterial hypertension with hyperuricemia. In view of the largest abundance in the blood of red blood cells, the state of their aggregation is most significant for providing an aggregate state of blood. The aim of the work is to assess the degree of disruption of aggregation properties of erythrocytes in patients with hypertension with hyperuricemia. 41 patients with arterial hypertension of 1-2 degree with hyperuricemia of the second adulthood were examined. The control consisted of 26 clinically healthy people of the second adulthood. Biochemical, hematological and statistical methods of investigation were used. In patients, an excess of cholesterol in erythrocyte membranes was found in the study, a decrease in the level of total phospholipids in them and activation of lipid peroxidation in them. In patients, an excess of spontaneous aggregation of erythrocytes was found. This should be considered as a consequence of the lipid peroxidation, metabolic disturbances, increased spasm of blood vessels and changes in the ratio of biologically active substances in the blood of hypertensive hyperuricemia. The presence of hyperaggregation of erythrocytes in patients with arterial hypertension with hyperuricemia dramatically increases the risk of thrombosis, which can lead to a permanent disability they are fatal. Keywords: arterial hypertension, hyperuricemia, pathology, aggregation, erythrocytes.



*Corresponding author

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INTRODUCTION

Until now, among the working population in developed countries, the prevalence of a combination of arterial hypertension (AH) and hyperuricemia persists [1,2]. The simultaneous presence of these two pathologies in a person greatly increases the probability of developing a thrombosis of any localization threatening disability and death [3,4].

The high incidence of thrombosis in patients suffering from hypertension and hyperuricemia simultaneously is due, undoubtedly, to the presence of hyperaggregation of blood cells in them [5,6], which is manifested by worsening of blood rheology. This situation strongly stimulates hemostasis and increases the risk of thrombosis [7,8,9]. It is known that the aggregation of blood cells is inhibited by substances by disaggregants synthesized in the vessel wall. Apparently, in this situation, the sensitivity of blood cells to disaggregants (prostacyclin and nitrogen oxide) decreases [10,11]. The wide prevalence of the combination of hypertension with hyperuricemia is of great interest to her, including the state of spontaneous aggregation of erythrocytes in this category of patients.

The aim of the work is to assess the degree of disruption of aggregation properties of erythrocytes in hypertensive patients with hyperuricemia.

MATERIAL AND METHODS

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

41 patients with AH of 1-2 degrees, risk 4 [12] with hyperuricemia of the second adulthood (mean age 54.4±2.4 years) were examined. The control group included 26 clinically healthy people of the second adulthood. All persons involved in the study gave written information consent to participate in it, according to the generally accepted procedure [13].

The state of lipid peroxidation (LPO) in plasma was determined by the level of thiobarbituric acid (TBA) - active products by the Agat-Med kit (Russia) and the number of acyl hydroperoxides (AGP). [14] The level of antioxidant capacity of plasma was determined by the method of [15].

The activity of LPO in erythrocytes was determined by the level of malonic dialdehyde (MDA) in them and the content of AGP in them after washing and resuspension [14]. Also, in the washed and resuspended red blood cells, the amount of cholesterol was determined by the enzymatic colorimetry method by the kit of the company Vital Diagnosticum (Russia) and the total phospholipids by the phosphorus content in them by the conventional method.

The level of spontaneous aggregation of erythrocytes in a plasma obtained without superimposing a tourniquet was recorded under a light microscope in Goryaev's chamber [16]. The values of the number of erythrocyte aggregates, the number of aggregates and erythrocytes not aggregated [17] were taken into account.

The results were processed by Student's criterion (t). Statistical processing of received information was made with the help of a programme package "Statistics for Windows v. 6.0", "MicrosoftExcel". Differences in data were considered reliable in case of p<0.05.

RESEARCH RESULTS AND DISCUSSION

In the examined patients, activation of LPO in plasma was found - the amount of AHP in it exceeded the control by 2.3 times, the concentration of TBA-active products - by 1.5 times, as a result of attenuation of the antioxidant activity of plasma by 1.5 times (Table).

The examined patients showed an increase in the content of cholesterol in the erythrocyte membranes with a decrease in total phospholipids in them. At the same time, in red blood cells, LPO was enhanced due to the weakening of their antioxidant defense (Table).



Spontaneous aggregation of erythrocytes was increased in all patients (Table). This was judged by an increase in the level of their total involvement in aggregates (by 62.7%), an increase in the number of these aggregates (by 50.0%) and a decrease of 59.2% in non-aggregated red blood cells.

Registrated parameters	Patients, n=41, M±m	Control,
		n=26 <i>,</i> M±m
acylhydroperoxides plasma,	3.27±0.08	1.42±0.09
D ₂₃₃ /1ml		p<0.01
TBA-compounds, umol / I	5.38±0.12	3.56±0.07
		p<0.01
antioxidant activity plasma, %	22.2±0.17	32.9±0.12
		p<0.01
biochemical p	arameters of erythrocytes	
cholesterol of erythrocytes, umol/10 ¹²	1.34±0.008	1.04±0.004
erythrocytes		p<0.01
common phospholipids of erythrocytes,	0.5±0.010	0.75±0.003
umol/10 ¹² erythrocytes		p<0.01
acylhydroperoxides of erythrocytes,	4.70±0.13	3.08±0.10
D ₂₃₃ /10 ¹² erythrocytes		p<0.01
malonic dialdehyde of erythrocytes,	1.65±0.07	1.14±0.05
nmol/10 ¹² erythrocytes		p<0.01
catalase of erythrocytes, ME/10 ¹²	7450.0±14.0	11196.0±22.4
erythrocytes		p<0.01
superoxidismutase of erythrocytes, ME/10 ¹²	1650.1±2.00	1986.0±7.01
erythrocytes		p<0.01
aggrega	tion of erythrocytes	
sum of all the erythrocytes in an aggregate	68.2±0.15	41.9±0.10
		p<0.01
quantity of aggregates	13.5±0.16	9.0±0.06
		p<0.01
quantity of free erythrocytes	150.7±1.02	240.0±0.23
		p<0.01

Table. Registered indicators in the surveyed

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

Great importance in the development of rheological disorders and the formation of a risk of thrombosis in patients with AH and hyperuricemia has an increase in erythrocyte aggregation [18, 19]. In patients with a combination of hypertension and hyperuricemia, the antioxidant activity of plasma decreases, which causes an increase in the activity of LPO in it [20]. This strongly damages the erythrocyte membranes [21] and is burdened by the development in the erythrocytes of these patients of the lipid imbalance that promotes their hyperaggregation. At the same time, the ability of red blood cells to disaggregate weakens [22,23]. The combination of these processes forms the growth of erythrocyte aggregation in the observed patients [24]. The increase in erythrocyte aggregation in hypertensive patients with hyperuricemia is largely due to the weakening of their disaggregating mechanisms [25,26] and to a decrease in the number of negative proteins on the erythrocyte membranes [27]. The onset of depression of the antioxidant properties of the plasma forms an intensification of peroxidation processes in it and as a result of it pronounced oxidative damages of endotheliocytes and globular plasma proteins arise [28,29]. In the prevailing conditions, the strengthening of erythrocyte communication among themselves is increasing, leading to an increase in the number of their aggregates [30, 31]. In this case, the depression of the sensitivity of erythrocytes to prostacyclin and nitrogen oxide forms an imbalance in the erythrocytes of the activity of adenylate cyclase and phosphodiesterase [32,33]. This contributes to a decrease in the level of cyclic adenosine monophosphate in their cytoplasm and

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an increase in the amount of free Ca^{2+} , which additionally strongly stimulates the aggregation of erythrocytes [34, 35].

CONCLUSION

For patients with arterial hypertension and hyperuricemia, a high incidence of thrombosis is characteristic. This circumstance was an important reason for examining this contingent of patients. During the research, it was revealed that in patients with arterial hypertension and hyperuricemia, the level of antioxidant protection of plasma decreases and the processes of lipid peroxidation, which negatively affects erythrocyte aggregation, increase in it. It was found that in patients with arterial hypertension and hyperuricemia, a reduction in the disaggregation capacity of erythrocytes was found. The emerging situation of increasing aggregation of erythrocytes forms a risk of future vascular complications.

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