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# Aggregation Of Thrombocytes In Patients With Arterial Hypertension And Impaired Glucose Tolerance.

**Medvedev IN*.**

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

**ABSTRACT**

It has long been noted in modern society the growth in the number of patients suffering from both arterial hypertension and impaired glucose tolerance. For these patients, a high incidence of thrombosis is associated with hyperaggregation of their blood cells. The goal is to evaluate the aggregation capacity of platelets in patients with arterial hypertension with impaired glucose tolerance. We examined 49 patients of the second adulthood (mean age 52.4±1.9 years) with arterial hypertension 1- 2 degrees, risk 4 with impaired glucose tolerance. 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 in the work. The high incidence of thrombosis in various locations with hypertension with impaired glucose tolerance is apparently closely related to the development of platelet hyperaggregation. A serious cause of its occurrence in conditions of a combination of arterial hypertension with impaired glucose tolerance is the weakening of the antioxidant protection of the plasma with the activation of the processes of lipid peroxidation in it. It was found that people with arterial hypertension and impaired glucose tolerance have an obvious weakening of platelet disaggregation. 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, impaired glucose tolerance, pathology, aggregation.

***Corresponding author***

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## INTRODUCTION

In developed countries, high prevalence of arterial hypertension (AH), combined with a violation of glucose tolerance [1,2]. This combination causes a great risk of development in these patients, especially in adulthood, the mass of blood vessel thrombosis leading to widespread disability and mortality [3]. It is noted that the emergence of thrombosis of any localization is always promoted by hyperaggregation of blood cells, which is now increasingly common [4]. The formation of this state is accompanied by a gradual increase in the process of aggregation of blood elements, which strongly stimulates various mechanisms of hemostasis, sometimes leading to thrombosis [5,6,7]. Against the backdrop of hyperaggregation of blood cells, there is often a marked weakening of their sensitivity to vascular substances-disaggregants, the most functionally significant of which are prostacyclin and nitric oxide [8,9]. In view of the prevalence of hypertension with the violation of glucose tolerance, it was very important to evaluate the state of platelet aggregation in these patients [10].

The goal is to assess the aggregation capacity of platelets in patients with hypertension with impaired glucose tolerance.

## MATERIAL AND METHODS

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

We examined 49 patients of the second mature age (mean age 51.2±2.7 years) with AH of the $1^{st}$-$2^{nd}$ degree [11] 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. 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 phospholipid (CPL) according to the content of phosphorus in them.

Aggregation of platelets (AP) 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 ($5.0\times10^{-6}$ M) and with combinations of ADP and epinephrine; ADP and collagen; epinephrine and collagen at the same concentrations in a platelet-rich plasma standardized for platelet counts of $200\times10^{9}$ platelets [16]. The level of intravascular aggregation of platelets was determined using a phase contrast microscope with the registration of the number of small, medium and large aggregates and the degree of involvement of platelets in them [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 2.3 times, TBA-active products – in 1.5 times, being accompanied by suppression of antioxidant plasma activity in 1.43 times (Table).

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**Table. Registered indicators in the surveyed**

| Registrated parameters | Patients, n=49, M±m | Control, n=26, M±m |
|---|---|---|
| acylhydroperoxides plasma, $D_{233}$ /1ml | 3.25±0.08 | 1.42±0.09 p<0.01 |
| TBA-compounds, mcmol/l | 5.27±0.15 | 3.56±0.07 p<0,01 |
| antioxidant activity plasma, % | 23.0±0.18 | 32.9±0.12 p<0.01 |
| biochemical parameters of platelets | | |
| cholesterol of platelets, mkmol/$10^9$ platelets | 1.07±0.011 | 0,67±0,005 p<0,01 |
| common phospholipids of platelets, mkmol/$10^9$ platelets | 0.35±0.009 | 0,49±0,004 p<0,01 |
| acylhydroperoxides of platelets, $D_{233}$/$10^9$ platelets | 3.40±0.08 | 2,20±0,04 p<0,01 |
| malonic dialdehyde of platelets, nmol/$10^9$ platelets | 1.32±0.12 | 0,68±0,02 p<0,01 |
| catalase of platelets, ME/$10^9$ platelets | 5100.0±23.62 | 9790,0±20,10 p<0,01 |
| superoxidismutase of platelets, ME/$10^9$ platelets | 1120.0±7.94 | 1650,0±3,00 p<0,01 |
| aggregation of platelets | | |
| aggregation with ADP, s | 25.9±0.12 | 41,0±0,12 p<0,01 |
| aggregation with collagen, s | 24.0±0.13 | 33,2±0,10 p<0,01 |
| aggregation with thrombin, s | 36.9±0.10 | 55,3±0,05 p<0,01 |
| aggregation with ristomycin, s | 28.1±0.07 | 45,2±0,06 p<0,01 |
| aggregation with epinephrine, s | 72.0±0.15 | 93,0±0,07 p<0,01 |
| aggregation with ADP and epinephrine, s | 21.2±0.14 | 34,5±0,04 p<0,01 |
| aggregation with ADP and collagen, s | 17.8±0.08 | 26,6±0,05 p<0,01 |
| aggregation with epinephrine and collagen, s | 13.4±0.14 | 29,2±0,12 p<0,01 |
| The number of platelets in the aggregates, % | 12.6±0.18 | 6,5±0,07 p<0,01 |
| Number of little aggregates (in 100 free thrombocytes) | 16.1±0.20 | 3,1±0,03 p<0,01 |
| Number of medium and large aggregates (in 100 free thrombocytes) | 1.54±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.

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 hypertension and impaired glucose tolerance, acceleration of development of AP with inductors and their combinations was revealed (Table). The most accelerated AP developed with collagen, a little later with ADP, even later with ristomycin, thrombin and adrenaline. The onset of AP with combinations of inductors was even more accelerated. The number of platelet aggregates and the level of platelet involvement in patients with hypertension and impaired glucose tolerance exceeded the control figures.

Important significance in the development of rheological disturbances and thrombophilia in persons with AH and impaired glucose tolerance belongs to aggregation increase of regular blood elements and especially – platelets [19,20]. At combination of AH 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. At the same time, the level of disaggregating abilities in platelets decreases [23,24].

The enhancement of platelet aggregation is caused not only by increased mechanization of AP, but also by the weakening of mechanisms of disaggregation [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 an increase in the sensitivity of platelets in the factor of Willebrand with an increase in 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 the strong activation of aggregation mechanisms inside the vessels [31, 32].

## CONCLUSION

The activity of platelet aggregation is extremely important for maintaining homeostasis in the body. A serious manifestation of its disorders is the activation of aggregation processes of platelets. This phenomenon is very common in any cardiac pathology, including arterial hypertension. This disease is often combined with a violation of glucose tolerance, which is caused by the need for assessing the disaggregation capacity of blood vessels in relation to platelets in this contingent of patients. It is established that in patients with arterial hypertension and impaired glucose tolerance, there is a pronounced increase in aggregation properties of platelets. This is in this contingent of patients a serious cause of activation of hemostasis and the development of their risk of thrombosis of any localization.

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