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PECULIARITIES OF PLATELET HEMOSTASIS CHANGES IN PATIENTS WITH ARTERIAL HYPERTENSION AND CORONARY ARTERY DISEASE, DEPENDING ON THE PRESENCE OF MICROALBUMINURIA

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Abstract

The activity of platelets and microalbuminuria can be considered as a reflection of endothelial function both in arterial hypertension and in other cardiovascular diseases. To assess the platelet hemostasis we examined 98 people: 14 apparently healthy (Group 1, control), 34 patients with arterial hypertension, the 2-nd stage, in combination with coronary artery disease with concomitant MAU (group 2), 50 patients with arterial hypertension, the 2-nd stage, in combination with coronary artery disease without concomitant MAU (3 Group). Analysis of induced platelet aggregation revealed decrease in ADP and AA-induced aggregation in the study groups of patients, which may be due to the treatment of this category of patients with acetylsalicylic acid, which has a direct effect on the AA-stimulated aggregation. Decrease of expression of ADP aggregation in patients of surveyed groups may be due to a receiving clopidogrel by patients. It is noteworthy that despite treatment with antiplatelet drugs, collagen and epinephrine-induced aggregation were not significantly different from the control group.

Key words: arterial hypertension, coronary artery disease, microalbuminuria, hemostasis, platelets aggregation.

Introduction

The kidney is one of the main targets of hypertensive process. Currently, there is a tendency of increase the number of patients with chronic kidney disease due to high blood pressure (arterial hypertension). At the same time, the progression of chronic kidney disease and decreased kidney function increases the risk of cardiovascular events [1].

Recent studies suggest that microalbuminuria (MAU) (30-299 mg / day) is a marker of renal vascular endothelial dysfunction and is regarded as a predictor of renal disease, the development of end-stage renal failure, cardiovascular complications and cardiovascular mortality [2].

There are data indicating the high prevalence of MAU in patients with arterial hypertension. According to an international survey i-SEARCH, majority of patients with hypertension has MAU - 53 to 71%, the highest levels of urinary protein excretion were recorded in patients with uncontrolled hypertension. According to P. Bramlage et al., MAU is observed in 30-40% of patients with hypertension, the likelihood of its detection is determined by the duration and degree of severity of hypertension.

In the current guidelines for the management of patients with hypertension, even clinically insignificant renal dysfunction presented as important indicators of risk stratification, and from this point of view, the presence of MAU or reduced glomerular filtration rate (less than 60 mL / min per 1.73 m2) is considered a high factor of cardiovascular risk. Thus even a moderate increase in creatinine level in blood serum (1.3-1.5 mg / dL in males and 1.2-1.4 mg / dL in women), and / or MAU (30-300 mg / day) - indicators of organ damage in patients with hypertension [3].

Well known, that state of various parts of the hemostatic system may also be reflection of endothelial dysfunction, especially those most actively regulated by endothelial factors. These include, in particular, platelet hemostasis, which has the closest relationship with the vascular endothelium.

It was found that the amplification of platelet aggregation activity dramatically increases the risk of thromboembolic complications, which are one of the causes of mortality in patients with

hypertension. As the disease progresses there is deterioration in all indicators of peripheral blood circulation system [4, 5].

If damaged vascular endothelium, including atherosclerosis, endothelial adhesion occurs outcrops of proteins and platelet activation. Some substances: thrombin, collagen, adenosine diphosphate (ADP), epinephrine, arachidonic acid (AA), thromboxane A2 etc. cause activation and following aggregation of platelets. There is a change in platelet form with the formation of pseudopodia and their adhesion-bonding under the influence of inducers of aggregation. Besides activated there is spontaneous aggregation of platelets caused by changes in the composition of the plasma (increasing number of biologically active substances in blood), and increased ability to form platelet aggregates.

So, the activity of platelets, and the MAU can be considered as a reflection of endothelial function both in arterial hypertension and in other cardiovascular disease. However, data of the relationship status of platelet hemostasis and severity of MAU are rather limited, even though the issue can be not only scientific but also practical interest for the determination of adequate approaches to the treatment of these patients.

Purpose. Assess the spontaneous and induced platelet aggregation in patients with arterial hypertension and coronary artery disease and to identify features of the aggregation activity of platelets in the presence of microalbuminuria.

Materials and methods. During the study we examined 98 people: 14 apparently healthy (Group 1, control), 34 patients with arterial hypertension, the 2-nd stage, in combination with coronary artery disease with concomitant MAU (group 2), 50 patients with arterial hypertension, the 2-nd stage, in combination with coronary artery disease without concomitant MAU (3 Group). The patient groups were comparable in age and sex. The investigation was carried out in the first day after hospitalization.

Platelet aggregation was assessed using laser agregatometry. As aggregation inducers were used - AA at a dose 1.0 mmol, ADP at a dose of 2.5 micromoles, collagen-1.25 mg / ml, adrenalin – 5 micromoles, according guideline [6-9].

Statistical data processing was performed using the methods of variation statistics, correlation analysis, implemented in the program Statistica 6.0 (StatSoft, USA). The significance of differences of average values of the indicators assessed by the Mann-Whitney and Student. Statistically significant differences were considered when $p<0,05$.

Research results

Comparison groups of patients received antiplatelet drugs, which could affect the specific indicators of platelet hemostasis, which we considered in the analysis of the data.

Analysis of induced platelet aggregation revealed decrease in ADP and AA-induced aggregation in the study groups of patients (Table 1), which may be due to the treatment of this category of patients with acetylsalicylic acid, which has a direct effect on the AA-stimulated aggregation.

Decrease of expression of ADP aggregation in patients of surveyed groups may be due to a receiving clopidogrel by patients. However, the distribution of patients who received acetylsalicylic acid and clopidogrel in the surveyed groups were similar and comparable to that provided a basis for further comparison of groups.

It is noteworthy that despite treatment with antiplatelet drugs, collagen and epinephrine-induced aggregation were not significantly different from the control group.

Thus, it should be noted that the presence of concomitant microalbuminuria prevents adequate decrease in AA-stimulated platelet aggregation that may be mapping of the less efficient of acetylsalicylic acid in these patients. We have not found statistically significant differences between the groups with and without microalbuminuria when applying other inducers of aggregation.

Table 1

Indicators of platelet functional activity in different patients groups, Me (25%;75%)

Indicator	Control Group (n=14)	2 Group (n=34)	3 Group (n=50)	p 2-3
Spontaneous aggregation degree, %	0,86 (0,48;1,14)	0,47 (0,37; 1,07)	0,81 (0,25; 4,21)	0,2279
ADP induced aggregation degree, %	34,85 (30,3; 42,6)	19,05 (10,6; 33,0)**	18,5 (5,64; 39,6) *	0,7205
AA induced aggregation degree, %	28,65 (22,0; 35,3)	14,64 (1,76; 26,1)**	2,05 (0,53; 7,59) ***	0,0485
Collagen induced aggregation degree, %	21,74 (16,18; 25,1)	24,1 (5,53; 34,7)	30,95 (10,75; 47,95)	0,2410
Adrenalin induced aggregation degree, %	18,3 (2,66; 34,8)	9,44 (3,01; 23,3)	10,9 (5,12; 20,8)	0,4278

Note: significance of changes against the Control Group: *p<0,05, **p<0,01, *** p<0,001

Conclusions

The presence of concomitant microalbuminuria associated with higher rates of AA-induced platelet aggregation, which may be an indication of less activity of acetylsalicylic acid in patients with arterial hypertension in combination with coronary artery disease in the presence of microalbuminuria.

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