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PLATELET AGGREGATION IN PATIENTS WITH CORONARY HEART DISEASE AND DEPRESSIVE DISORDERS

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ABSTRACT: Platelets play an important role in the process of stopping bleeding in case of damage to the vascular wall and tissues, as well as in the process of intravascular thrombosis. During the implementation of primary platelet-vascular hemostasis (TSH), phases of reactions aimed at the formation of a blood clot are distinguished: adhesion, spreading, release reaction, aggregation and retraction. An important indicator in assessing TSH is the nature of changes in platelet aggregation under the influence of various inducers. Aggregation is rapid and irreversible due to the action of thrombin, adrenaline, adenosine diphosphate (ADP) and ristomycin in certain concentrations. The most reliable methods for assessing TSH are to study the rate and degree of decrease in the optical density of platelet plasma with the addition of aggregation inducers. Platelet aggregation under the action of ADP at a concentration (2.5 mmol/l) and ristomycin (10 mg/ml). ADP-induced aggregation makes it possible to identify a risk group for thrombosis, monitor treatment with disaggregants and confirm the presence of thrombasthenia. The use of this concentration of ADP contributes to the development of complete irreversible aggregation and the lag phase is not observed, and ristomycin-induced aggregation allows us to study the effect of the Willebrandt factor. The aim of the study was to study adenosine diphosphate (ADP)-induced platelet aggregation in patients with coronary heart disease (CHD) with anxiety and depression. It was found that the degree and time of platelet aggregation in individuals with coronary heart disease in combination with anxiety and/or depression were less than in patients with coronary heart disease without mood disorders. The aggregation time was significantly lower in the case of using ADP at a concentration of 1.25 and 2.5 microns. The degree of aggregation was significantly lower in the case of an ADP concentration of 1.25 microns. The results obtained do not support the hypothesis of increased thrombosis in patients with coronary heart disease with anxiety and depressive disorders.

Key words: coronary heart disease, depression, anxiety, platelet aggregation.

INTRODUCTION

The combination of coronary heart disease (CHD) and affective disorders of the anxiety-depressive spectrum is becoming more common and increases the mortality rate of such patients by 2-4 times. Currently, it is not completely clear what underlies this phenomenon. Increased thrombosis in patients with depression is considered as one of the possible causes discussed in the literature. Major depression is associated with dysfunction of serotonergic neurotransmission, while serotonin and serotonin or hydroxytryptamine (5NT) receptors are involved in biological the base of depressive disorders. A number of studies have shown that serotonin promotes endotheliocyte proliferation indirectly, possibly through (5–HT)2A receptors and with the participation of these same receptors can cause vasoconstriction. As a result of the similarity of physiological properties (5–HT)2A platelet receptors and serotonin-ergic neurons platelets are a peripheral model of neuronal synapses of the brain.

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At the same time, changes in (5–HT)2A receptor apparatus can initiate atherogenic and prothrombotic mechanisms at the periphery. For evaluation of thrombosis is widely used to study spontaneous and induced platelet aggregation. Such studies were conducted in patients with depressive disorders. However, there is practically no work on the functional activity of platelets in patients suffering from coronary heart disease and anxiety-depressive disorders at the same time. Therefore, the purpose of this study was to study the parameters of adenosine diphosphate (ADP)-induced platelet aggregation in patients with coronary heart disease in combination with affective disorders of the anxiety-depressive spectrum.

MATERIALS AND METHODS OF RESEARCH

70 patients (50 men and 20 women) aged 38 to 76 years (average age was 61.6 ± 8.4 years) suffering from stable forms were examined coronary heart disease: angina pectoris of various functional classes and postinfarction cardiosclerosis. All patients were tested using the Hospital Anxiety and Depression Scale. Based on these results, a number of patients underwent a psychopathological examination with the participation of a psychiatrist using the Hamilton anxiety and depression scales. The study did not include patients with unstable angina pectoris, functional class II–IV heart failure, malignant hypertension, hemodynamically significant heart defects, diabetes mellitus, systemic and oncological diseases, acute cerebrovascular accident and a history of traumatic brain injuries, myocardial infarction suffered less than 6 months before inclusion in the study research, as well as people suffering from alcoholism. In addition, the study did not include people taking anticoagulants. All patients signed an informed consent to participate in the study. Blood sampling was performed on an empty stomach, between 9 and 10 a.m. During the 3 days preceding the study, the patients did not take the disaggregants. ADP-induced platelet aggregation was evaluated on the aggregometer AR 2110 by the light scattering method according to the Born principle. ADP in concentrations of 0.625 microns, 1.25 microns, 2.5 microns and 5 microns was used as an inducer. The time and degree of aggregation were estimated. Statistical processing of the results was carried out using the statistical package SPSS 14.0. The data are presented as an average value with a standard error of the mean. The significance of the differences was assessed using Wilcoxon tests and Manna-Whitney.

THE RESULTS AND THEIR DISCUSSION

Based on the psychopathological examination data, all patients were divided into 2 groups. The first group consisted of 40 patients with coronary heart disease and with clinically significant anxiety-depressive disorders, the second (control group) consisted of 30 patients with coronary heart disease without affective disorders.There were no significant statistical differences in the main clinical characteristics in patients of different groups. Analysis of ADP-induced platelet aggregation showed that when exposed to ADP in all concentrations used in the group of CHD patients with anxiety and depressive disorders, the degree and time platelet aggregations were lower than in the control group. In this case, in the case of an inductor concentration the aggregation time was significantly lower at 1.25 and 2.5 microns ($p < 0.05$), and in the case of a concentration of 1.25 microns, the degree of aggregation was significantly lower ($p < 0.05$). The data we obtained do not support the hypothesis that patients with coronary heart disease suffering from anxiety and depressive disorders have a greater tendency to thrombosis than patients with coronary heart disease who do not have mood disorders. The results of the study showed that the degree and time of platelet

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aggregation in individuals with coronary heart disease in combination with there were fewer anxiety-depressive disorders than in patients with coronary heart disease without mood disorders. And if a decrease in the aggregation time in patients in the main study group correlates with the hypothesis under study, then a decrease in a more important indicator — the degree of aggregation — contradicts this postulate. There is a point of view that this method does not accurately assess changes in the functional activity of platelets in this group of patients. It was assumed that with the introduction of flow cytometry technology, it would be possible to more fully test the developing changes. However, far from all studies using flow cytometry, the results indicated an increase in aggregation rates in patients with depression. The level of serotonin, in turn, may be related to the peculiarities of functioning (5 HT)2A platelet receptors. A number of authors believe that 5NT2 And platelet receptors in patients with depression are functionally subnormal in the active phase of the disease, and this can also explain the decrease in platelet aggregation ability in patients with coronary heart disease and affective disorders. Finally, it is not completely clear how comorbid anxiety affects the indicators of induced aggregation in patients with depression. It is known that anxiety and depressive disorders are often combined, it is not always possible to draw a line between these nosological forms. In our study, both anxiety and depressive disorders were also observed in most patients.

CONCLUSIONS

Thus, when comparing the parameters of ADP-induced platelet aggregation in patients with coronary heart disease depending on the presence or absence of effective disorders of the anxiety-depressive spectrum, results were obtained that were not associated with increased thrombosis in patients with comorbid pathology. A decrease in the degree of aggregation in people with coronary heart disease suffering from anxiety and/or depression is an interesting phenomenon that requires further research in this area.

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