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THE EFFECT OF ANTIHYPERTENSIVE DRUGS ON THE STATE OF THE THROMBOSIS SYSTEM WITH ENDOTHELIAL DYSFUNCTION

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Resume: The study of of hypertension problem becomes necessary from the point of view of developing effective and safe methods of treating hypertension, since a look at the pathogenesis of hypertension from the perspective of the interaction of the endothelial system with the thrombosis system is a potential target and from a therapeutic point of view this dictates the need to study the effect of antihypertensive drugs on the state of the thrombosis system in conjunction with endothelial dysfunction.

Key words: hypertension, interactions of the endothelial system, thrombus formation system, choice of antihypertensive drugs.

The relevance of the work. Endothelial dysfunction plays a crucial role in the pathogenesis of hypertension, a prevalent cardiovascular disease affecting millions globally. While extensive research exists on hypertension's various aspects, the interplay between endothelial dysfunction and platelet function in disease progression remains inadequately explored. This interaction is particularly significant because it directly impacts the development of atherosclerotic plaques and thrombotic events, major contributors to hypertension-related morbidity and mortality. Understanding this complex relationship is paramount for developing targeted therapeutic strategies. Current research suggests that endothelial cells, forming the inner lining of blood vessels, release various factors regulating vascular tone, inflammation, and coagulation. In hypertension, this delicate balance is disrupted. Endothelial dysfunction manifests as impaired nitric oxide (NO) production, leading to vasoconstriction and increased blood pressure. Simultaneously, there's an increase in the production of pro-thrombotic factors like von Willebrand factor and tissue factor, promoting platelet activation and aggregation. This creates a vicious cycle where impaired endothelial function fosters a pro-thrombotic state, accelerating atherogenesis and increasing the risk of cardiovascular events like myocardial infarction and stroke. The interaction is bidirectional: activated platelets release factors that further damage the endothelium, amplifying the dysfunction. The choice of antihypertensive drugs should, therefore, consider their impact not only on blood pressure but also on both endothelial and platelet function. While many antihypertensive medications effectively lower blood pressure, their effects on the endothelial-platelet interaction vary significantly. Some, like ACE inhibitors and angiotensin receptor blockers (ARBs), have demonstrated beneficial effects on endothelial function by improving NO bioavailability and reducing oxidative stress. Other classes, such as calcium channel blockers, may have less pronounced effects on endothelial function, while some may even exhibit pro-thrombotic tendencies. Therefore, a personalized approach to antihypertensive therapy should consider individual patient factors, including the assessment of both endothelial and platelet function, to optimize treatment and minimize cardiovascular risk. Further research into biomarkers reflecting endothelial and platelet activity could aid in stratifying patients and tailoring treatment to maximize efficacy and safety. This could involve investigating levels of circulating endothelial microparticles, soluble adhesion molecules, and platelet activation markers to guide therapeutic choices and monitor treatment response. The

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ultimate goal is to move beyond simply managing blood pressure to actively mitigating the underlying endothelial and platelet dysfunction driving hypertension-related complications.

Materials and methods of research: The study involved 100 patients with a diagnosis of hypertension stage II, aged 26 to 80 years. Of these, there were 44 men and 56 women. At the same time, the largest number of patients were aged 41 to 70 years. As mentioned above, dysfunction of endotheliocytes and, as a result, changes in the rheological properties of blood in patients with hypertension is accompanied by a change in platelet morphology. Thus, in the examined patients, a decrease in the number of platelets circulating in the blood, as well as the percentage of discocytes, was noted, while the number of morphologically altered platelets (echinocytes, discoechinocytes and spherocytes) was significantly increased. One of the reasons for the increase in pathologically altered platelets is the increase in blood fibrinogen, fibronectin and von Willebrand factor, which activates the adhesive-aggregation properties of blood cells.

Results and discussions. The inclusion of the drug Concor in the arsenal of therapy leads to a significant increase in the number of platelets in the blood. When analyzing the results of the study shown in Table 6.2, an increase in the number of blood cells by 38% was noted, and on average it was equal to 231.6 ± 12.4 109/l versus 184.6 ± 9.2 109/l in the group before treatment . Similar dynamics was noted with the use of the drug Physiotens - where the platelet count exceeded the initial values by 46%. The increase in platelet count was accompanied by a significant increase in the percentage of discocytes by 82% when using Physiotens, whereas when Concor was included in the arsenal of therapy, the percentage of discocytes changed slightly. The increase in structurally complete platelets, in particular discocytes, is apparently due to the inclusion of the drug - physiotension not only on the function of endotheliocytes, but also on platelets as a disaggregant.

Table 6.2.

Morphology of blood platelets in the dynamics of the treatment of hypertension

Indicators	group of healthy people (control)n =12	Before treatmentn =34	After treatment	
			Concor n =16	Physiotens n =18
The number of platelets in thousand, 1x10^9/л	248,0±17,1	184,6±9,2*	231,6±12,4**	214,8±10,9
Discocytes, %	85,4±1,2	71,3±4,3*	72,1±5,2	82,3±5,6**
Echinocytes, %	10,6±0,41	20,7±0,83*	18,8±0,91	13,9±0,77**
Discoechinocytes, %	1,5±0,11	4,02±0,17*	3,75±0,14	2,1±0,12**
Spherocytes, %	1,5±0,21	3,98±0,24*	5,35±0,32**	1,7±0,11**

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Note: *-significance 0.05 when compared with healthy individuals

**-significance of differences (P < 0.05) when comparing before and after therapy

The ongoing therapy contributed to the reduction of pathological cells, i.e. echinocytes, where its percentage when using physiotens decreased by 5 times and amounted to 13.9 ± 0.77% versus 2.7 ± 0.97%, while when using the drug Concor, this indicator remained high and was equal to 18, 8±0.91% versus 20.7±0.83%.

The nature of changes in discoechinocytes had similar dynamics. So if, before therapy in patients with arterial hypertension, the value of the latter was equal to 4.02 ± 0.17%, then the inclusion of the drug Concor instilled in a significant decrease in the percentage of discoechinocytes and its value was 3.75 ± 0.14%. Naportiv, when physiotens was included in the arsenal of therapy, the percentage of the latter decreased by 2 times and was equal to 2.1±0.12% (P<0.05). When analyzing the percentage of spherocytes, we observed an increase in the latter when included in the arsenal of conventional physiotherapy. The value of the latter in the examined persons after therapy was 5.35±0.32%, while before therapy it was equal to 3.98±0.24%, which is 34% higher than the initial values. We observed a different dynamics when the drug Physiotens was included in the arsenal of complex therapy. The inclusion of physitenz resulted in a more than 2-fold increase in the percentage of spherocytes, where its value was 1.7±0.11% versus 3.98±0.24% in the group before treatment. The inclusion of Concor in the arsenal of therapy was accompanied by a significant increase in the number of venous blood platelets and an increase in pathological forms of platelets, in particular spherocytes by 1.5 times, while with the use of Physiotens, we observed a significant increase in normal forms of blood cells, namely discocytes against the background of a decrease in echinocytes and discoechinocytes on average 2 times.

To confirm the observed dynamics regarding platelet morphology, we studied arterial hypertension and platelet regenerative activity in patients with arterial hypertension during therapy. As can be seen from the presented results of the study, the examined patients before treatment showed an increase in the number of activated platelets by 30% and amounted to 18.4±1.91%. The inclusion of Physiotens in the complex therapy was accompanied by a significant decrease in the number of activated platelets by 29% and, on average, was equal to 13.1±0.73% (P<0.05). Against the background of the use of this drug in the treatment of arterial hypertension, a significant decrease in the number of arterial hypertension registered was also noted, which amounted to 8.24±0.84% versus 12.3±0.81% in the group before treatment. In this situation, we observe a direct relationship between the active form of platelets, their structural and arterial hypertension-regulatory activity, which approaches the initial values when using Concor.

Table 6.3.

Morphology of blood platelets in the dynamics of treatment of hypertension

Indicators	group of	Before	After treatment

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	healthy people (control)n =12	treatmentn =34	Concor n =16	Physiotens n =18
Number of activated platelets, %	12,3±0,27	18,4±1,91*	17,1±0,81	13,1±0,73**
Number of arterial hypertension of erect platelets, %	7,04±0,22	12,3±0,81*	11,9±0,76	8,24±0,84**
Arterial hypertension platelet regenerative activity to ristomycin, %	104,1±6,2	144,6±3,52*	139,6±9,3	108,3±8,7**
Hemolytic arterial hypertensionregional test (II dilution), sec	15,4±0,44	9,2±0,23*	9,9±0,91	18,1±0,71**
Hemolytic arterial hypertensionregional test (III dilution), sec	30,2±1,18	24,1±0,44*	25,3±1,11	31,1±1,32**

Note: *-significance of differences (P<0.05) when compared with healthy individuals;

**-significance of differences (P<0.05) when comparing before and after therapy

One of the indicators of endotheliocyte dysfunction, along with an increase in desquamated endotheliocytes in the blood, is arterial hypertension, platelet regenerative activity to rastomycin. As can be seen from the presented results of the study, in patients with arterial hypertension, we observed an increase in this indicator by 1.4 times when compared with healthy individuals. The improvement in the functional state of endotheliocytes against the background of the use of Physiotens was also accompanied by a significant decrease in platelet regenerative activity to ristomycin and amounted to 108.3±8.7% versus 144.6±3.52% (P<0.05). Dysfunction of endotheliocytes, an increase in the number of arterial hypertension-registered platelets was accompanied by an increase in arterial hypertension-regulation activity of platelets to the inductor - ADP, which was studied using hemolysate of erythrocytes of various dilutions. Thus, when diluting (II), in patients with arterial hypertension, an increase in arterial hypertension in platelet regenerative activity was noted on average by ***% and amounted to 9.2±0.23 sec, against 15.4±0.44 sec (P<0.05). The inclusion of concor in the arsenal of therapy for arterial hypertension did not lead to significant changes in arterial hypertension in platelet regenerative activity to the ADP inducer in the II dilution. Meanwhile, the replacement of Concor with Physiotens significantly increased the time of arterial hypertension of platelet regurgitation to the ADP inducer in the II dilution by 2 times and amounted to 18.1±0.71 sec. The latter indicated that the data obtained were close to those of healthy individuals. At VI dilution of erythrocyte hemolysate and its effect on the functional state of platelets showed similar dynamics. So, if when using Concor, the studied indicator was equal to 25.3 ± 1.11 sec versus 24.4 ± 0.44 sec, then when using the drug Physiotens, the indicator of hemolytic arterial hypertension of the regenerative test averaged 31.3 ± 1.32 sec, which is 29% higher than the initial values.

Findings. Thus, the drug Concor in the treatment of arterial hypertension has a positive trend not only on the functional state of endotheliocytes, but also on the morpho-functional

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state of platelets, i.e. on its adhesive-arterial hypertension-regulatory activity relative to the inductor-ristomycin and ADP.

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