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AN ADDITION TO THE MOLECULAR MECHANISM OF HYPERTENSION DEVELOPMENT

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Abstract: The lack of effective treatment options for hypertension indicates that the pathogenetic elements of this disease are not yet fully understood. Therefore, the study and clarification of the pathogenesis of hypertension is an urgent task of modern medicine.

Key words: atherosclerotic vascular stenosis, hypoxia, anaerobic and aerobic glycolysis.

Introduction. Nowadays certain successes have been achieved in studying the mechanisms of hypertensive disease (HD) development, on the basis of which new ways of treatment of this disease have been developed [1,3,11].

However, the practice of using existing methods of treatment of HB has shown that the use of even the most modern drugs reduces blood pressure (BP) for a certain period, that is, after the action of this vasodilator drug BP rises again. It means that in clinical medicine there are not yet effective ways of GB treatment, under the action of which the molecular mechanisms of GB development would be eliminated. Low efficiency of existing methods of GB treatment has led to the increased incidence and development of various complications in the form of acute and chronic disorders of cerebral circulation [5,6,12]. In addition, the results of clinical observations show that not only the incidence rates have been increasing recently, but also the rejuvenation of GB [7,8,13].

Based on the above, it can be assumed that the molecular mechanisms of GB development are poorly understood. So, determination of new elements of etiology and molecular mechanisms of GB development, allow to develop new effective ways of treatment. Therefore, the study and clarification of the etiology and molecular mechanism of GB development is an urgent problem of modern medicine[9,10,14].

Purpose of the study: To investigate the molecular mechanism of GB development.

Material and methods of research. The study was carried out in 87 (48 women and 39 men) patients with GB: first stage 8 - patients; second stage 47 - patients and 3rd stage 23 - patients. The age of patients ranged from 30 to 60 years. On average 53 +- 5.0. Duration of the disease from 3 to 10 years (average 5.4 +- 0.5). Diagnosis of GB was carried out by dynamic tonometry and on the basis of data of clinical and neurological studies. To determine the morphological changes developed in the brain, all patients underwent magnetic resonance imaging (MRI) of the brain (1.5 tesla). The intensity of cerebral blood flow (the intensity of oxygen supply to the brain tissue) was judged by the value of systolic blood flow velocity in large cerebral vessels, which was determined by transcranial Dopplerography (TCDG). The latter was performed in all patients (13).

To determine the causes of cerebral blood flow insufficiency development, MR angiography of intracranial and brachiocephalic arteries was performed in all patients (23).

Proceeding from the fact that all biology, physiology and morphology of the human organism are based on adequate intracellular energy synthesis (1,7-13) and its intensity depends on the amount of incoming oxygen, we decided to study the state of cerebral blood flow and intracellular energy synthesis in the brain substance in GB patients.

The intensity of metabolism in the brain tissue was judged by the value of acid-alkaline balance (AAB) and residual oxidisability of cerebrospinal fluid (R.O.S.F). A lumbar puncture at a typical site was performed on all patients to determine CSF and residual cerebrospinal fluid oxidisability. CSF of cerebrospinal fluid was determined using a household PH-meter (FRG), and R.S.G was determined according to the method of K.S. Kosyakov (7). Statistical processing of the obtained data was performed using descriptive methods and ANOVA model. Assessment of the change in the indicators in comparison with the baseline was carried out using t - test.

Results: Clinical signs of GB corresponding to the stages of development of this disease were observed in all patients. The results of dynamic measurement of A/D showed that all patients had persistent A/D increase. MRI - tomograms of the brain showed the following:

a) in patients with stage 1 GB (8 - patients) moderate dilation of the subarachnoid space of the convexital surface and paravasal canals of the brain was noted:

b) patients with stage 2 GB on MRI showed marked dilation of the subarachnoid space of the convexital surface, paravasal canals and cerebral base cisterns with moderate hydrocephalus:

c) in patients with stage 3 GB, MRI of the brain showed marked enlargement of subarachnoid space of the convexital surface, paravasal canals and cisterns of the brain base with marked enlargement of the ventricular system of the brain.

Thus, the study of MR - tomographic data showed that in all patients with GB the phenomenon of encephalopathy was observed. The severity of encephalopathy depended on the stage of GB.

The results of ultrasound determination of systolic blood flow velocity (TCDG) showed that: in patients with the first stage of the disease systolic cerebral blood flow velocity was up to 120cm/sec, in patients with the second stage of GB was from 120cm/sec to 200cm/sec.(Average 170+-30), and in patients with the third stage of the disease systolic cerebral blood flow velocity was more than 200cm/sec. (Mean 230+-10). The results of TCDG studies showed that in all patients with GB, depending on the stage, acceleration of systolic blood flow velocity was observed, which resulted in a significant decrease in oxygen penetration from the vascular channel into the brain tissue. Thus, all patients with GB had cerebral circulatory disturbances of different severity.

The results of contrast-free MR - angiography showed that:

a) In patients with the first stage of the disease angiographically noted atherosclerotic stenosis of the initial section of vertebral arteries;

b) in patients with the second stage of the disease MR angiographically marked atherosclerotic stenosis of vertebral arteries was more pronounced in the initial and suboxipital part of the vessels.

Total atherosclerotic narrowing of the arterial lumen in the carotid artery basin was more pronounced in the area of bifurcation and initial parts of the internal carotid arteries;

c) in patients with the third stage of the disease, MR-angiograms showed marked atherosclerotic stenosis of intracranial and brachiocephalic arteries.

When performing lumbar puncture in a sitting position in all patients, colourless, clear fluid was obtained, flowing out in frequent drops. The magnitude of the liquor pressure averaged 360+-10 water column. 5 - 6 ml of fluid was released for determination of CSF and OOCSJ.

In patients with stage I disease, the CSF averaged 6.2+-0.5. In patients of the second group was 5.8+-1, and in the third stage of the disease was 5,.4+-2.0.

The values of residual cerebrospinal fluid oxidisability (ROC.C.S.J) in patients with stage I disease was 15.7+ - 5mg/%, in patients with stage II disease was 27+-2..0mg%, and in patients with stage III disease was 31+-2.0ml% (mean).

Discussion

Presence of MRI signs indicating reduction of brain volume and mass, reduction of oxygen supply to the brain as a result of development of atherosclerotic lesion (atherosclerotic stenosis) of vertebral arteries show that GB is not a functional disease (neurosis of the vascular centre) of the central nervous system, but is an organic disease of the central nervous system accompanied by brain atrophy (reduction of brain volume and mass) - dyscirculatory encephalopathy.

Acceleration of systolic velocity of cerebral blood flow in cerebral arteries (on TCDG) and the presence of atherosclerotic stenosis of vertebral and carotid arteries (on MR angiograms of intracranial and brachio-cephalic arteries) shows that the development of atherostenosis of vertebral and carotid arteries leads to the development of hypoxia in the area of the vascular centre (bottom of the rhomboid fossa). As a result of hypoxic irritation of the floor of the rhomboid fossa, the tone of peripheral arteries increases, i.e. the A/D rises. As a consequence of the latter, oxygen supply to the brain tissue increases.

It has been found that to maintain the vital activity of some cells is enough to maintain a small amount of energy, and other cells need a large amount of energy. Thus, the brain, which is fed only by carbohydrates, in a state of rest "eats" 60% of glucose and 20% of oxygen brought by blood into the body, because to maintain the higher nervous (cognitive) functions of the brain requires a very large amount of energy (ATP).And if we talk about the higher nervous (cognitive) functions of the brain is more energy-consuming process and to think of a more energy-consuming process is unimaginable. The brain does not create any reserves of energy substrates, incoming substrates (glucose) and oxidant (oxygen) are immediately spent on active work. Increase of acidity and residual oxidability of cerebrospinal fluid in our patients shows that the energy formed as a result of insufficient supply of glucose and oxygen to brain tissue develops glycolytic metabolism, which is extremely insufficient to maintain the vital activity of brain cells. As a result of energy insufficiency, a part of brain cells dies (apoptosis of nerve cells). And a part of cells begins to exist at the expense of exothermic energy, formed as a result of step-by-step cleavage of macromolecules (protein-mucopolysaccharide complex) of matrix into smaller molecules (to deaminated amino acids and glucose) - catabolic, destructive metabolism. However, the amount of energy generated by catabolic metabolism satisfies only relatively short-term cellular needs. Therefore, catabolic metabolism in damaged brain tissue progresses imperceptibly. (1,9-13). In addition, depression of intracellular energy synthesis is always accompanied by activation of the release of highly reactive free radicals (HRFRs) and oxygen intermediators that are highly destructive - oxidative stress (9-10).

The above shows that as a result of aggravation of catabolic metabolism and oxidative stress atrophic processes- encephalopathy develops in brain tissue.

The results of comparative analysis of brain MRI data, TGDG data, MR angiography and residual oxidability of cerebrospinal fluid showed that the values of A/D increase are proportional to the value of oxygen deficiency. So energy insufficiency developing due to insufficient oxygen supply to the brain tissue is the main mechanism of GB development and the phenomenon of encephalopathy. The latter developing due to hypometabolism in brain tissue is the main morphological element of GB.

On the basis of our own and literature data we can make the following.

Conclusion: the main mechanism of GB development is oxygen deficiency in brain tissue developing due to atherosclerotic stenosis of vertebral and carotid arteries;

encephalopathy developing due to catabolic metabolism and oxidative stress is the main morphological element of GB.

Atherosclerotic stenosis of vertebral or carotid arteries in varying degrees of severity is the main cause of the development of oxygen deficiency in brain tissue.

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