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Modern Approach to Diagnosing Cerebrovascular Diseases

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Abstract: Patients with NCF caused by cerebrovascular pathology are characterized by a decrease in mental activity and the speed of information processing, as well as a relatively milder severity of disorders of some higher brain functions (aphasia, agnosia, apraxia). The diagnosis is based on determining the vascular nature of the process, identifying the NCF syndrome itself and assessing its severity. Treatment of patients with NCF with chronic cerebrovascular accidents includes the correction of modifiable cardiovascular risk factors and the use of drugs that improve metabolism in the brain. One of these drugs is Cytoflavin, which has a significant neuroprotective effect. Data are presented on the effectiveness of Cytoflavin in patients with various forms of chronic cerebrovascular diseases. In such patients, a positive effect of the drug on the state of cognitive functions has been proven.

Keywords: dyscirculatory encephalopathy, chronic cerebral ischemia, asymptomatic cerebral infarction, cognitive impairment, Cytoflavin.

Ischemic brain lesions are represented by a wide range of syndromes with different mechanisms of development and clinical manifestations. All these syndromes are united by the main link in pathogenesis - the mismatch between the amount of oxygen and glucose entering the body and the needs of brain tissue. Glucose and oxygen deficiency leads to a violation of the ionic balance, inhibition of energy metabolism and the synthesis of proteins, neurotransmitters and other substances necessary for the normal functioning of nervous tissue. Currently, many stages of early and late ischemic damage to brain tissue are well studied [5].

One of the main criteria for classifying ischemic brain lesions is the rate of development of ischemia and neurological deficit. A stroke (stroke, TIA) is a rapid cessation of blood flow to the brain tissue. Its main causes are atherothrombosis, cardiogenic or arterio-arterial embolism, occlusion of small cerebral arteries. It is quite reasonable that the term "chronic cerebral ischemia" (CHI) was proposed to describe conditions characterized by a gradual (or relatively gradual) increase in the severity of clinical manifestations.

The risk of developing CICM due to possible progressive damage to the medulla under conditions of reduced blood flow has been confirmed by experimental studies [20]. In the case of insufficient blood supply, primary damage to the white matter of the cerebral hemispheres has been shown. An ischemic pathobiochemical cascade plays an important role in the pathogenesis of CICM, including impaired neurotrophic supply of neurons, slowing down the formation of synapses and inhibiting their strengthening, reducing the sensitivity of receptors to neurotransmitters, induction of apoptosis, etc. To a large extent, the progressive damage to the brain substance in CICM is also facilitated by the development of a neurodegenerative process with the accumulation of β-amyloid characteristic of Alzheimer's disease.

In this regard, common risk factors for cerebrovascular diseases, in particular CICI and Alzheimer's disease (hypertension, diabetes mellitus), attract attention, which indicates the commonality of the mechanisms of formation of these diseases. A characteristic morphological manifestation of CIGM is

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leukoaraiosis, which primarily indicates progressive changes in the white matter of the cerebral hemispheres due to impaired arterial blood flow.

However, “chronic” brain damage is not always truly chronic. Lacunar and spongy states resulting from damage to small-caliber cerebral arteries (diameter from 0.3 to 1.0 mm) in hypertension and some other pathological conditions are well known. Since small cerebral arteries are the last type of vessels that do not anastomose with each other, a decrease in blood flow in them leads to small lacunar infarcts. The development of microangiopathy is accompanied by the development of new infarcts with diffuse damage to the medulla. Various mechanisms of such damage include direct development of lacunar infarcts and degenerative damage, in particular, due to Turck-Waller degeneration, with subsequent cyst formation or replacement of the damaged area with glial tissue. Neuroimaging studies (MRI, CT) reveal post-ischemic foci in the brain tissue of such patients.

Clinically, the course of chronic cerebrovascular pathology with recurrent lacunar infarctions is characterized by episodes of sharp deterioration of the patient's condition, accompanied by an exacerbation of neurological deficit. After an episode of exacerbation, it is possible to stabilize the condition or even reduce the severity of the disorders due to the introduction of compensatory mechanisms, although in general the course of the disease is progressive. In the domestic literature, the combination of chronic ischemic damage to the brain substance with episodes of acute cerebral ischemia is considered dyscirculatory encephalopathy, which more accurately characterizes the nature of the pathological process than the concept of CIGM. It is worth noting the unequal meaning of these terms: despite their proximity, when using one of them instead of the other, the pathological condition of a particular patient may not be fully clear. The term CIGM is more consistent with the progressive course of the disease without acute cerebrovascular accidents and describes a condition that may be one of the components of dyscirculatory encephalopathy. On the other hand, both concepts indicate the leading role of vascular damage to the brain in the development of the pathological process, their use helps to choose an adequate therapeutic strategy;

The difficulty in distinguishing between acute and chronic cerebrovascular diseases is also associated with the possibility of so-called silent or asymptomatic cerebral infarction. Since the pathological focus is located outside the functionally important area of the brain, the disease may not be accompanied by focal neurological deficits or may not attract the attention of the patient due to its insignificant severity. Despite its relatively mild course, asymptomatic cerebral infarction is associated with an increased risk of recurrent cerebrovascular accidents, as well as the onset of cognitive impairment (CDI) and its progression to the formation of severe dementia [6].

The most common and characteristic manifestations of chronic cerebrovascular pathology are NKF and affective disorders. As a rule, they develop gradually and pass through the stage of non-dementia NCF, including mild and moderate forms [19]. It is very important that NCF, which does not significantly affect daily activities and is confirmed only by neuropsychological tests, is noticed by the patient himself or his relatives. Perhaps, the timely detection of the disease at this stage and the initiation of adequate therapy will help to delay the onset of severe cognitive impairment.

The clinical picture of NCF in patients with chronic cerebrovascular diseases is primarily associated with the formation of secondary dysfunction of the frontal lobes of the brain, which is associated with a violation of the connections between the anterior cortical and subcortical sections. NCF is based on a decrease in the activity of mental processes, difficulties in switching cognitive programs, and insufficient control over current cognitive activity [8]. Difficulties in all cognitive areas are characteristic, but impaired attention, a decrease in the speed of intellectual operations, impaired spatial praxis, and gnosis are most pronounced. Memory impairment, as a rule, is of a secondary nature and is caused by insufficient activity and selectivity of reproduction with relative safety of memorization and storage of information. In some patients, memory impairment is especially pronounced and prevails over other functional disorders, which allows them to be considered as an amnestic type of NCF, which is associated with a high risk of developing Alzheimer's or mixed dementia in the future [7, 15].

As the disease progresses, the level of NCF increases, reaching dementia, in which there is a pronounced diffuse impairment of all cognitive functions, as a result of which the patient loses independence and autonomy in everyday life [9]. In this situation, NCF has the characteristics of both vascular and Alzheimer's dementia, differential diagnosis based only on the clinical features of the disease is difficult, and even a clear identification of one or another type of dementia does not always help to choose an effective therapy.

Affective disorders in patients with chronic cerebrovascular diseases are usually manifested by vascular depression and emotional lability [4]. Such patients are characterized by mild to moderate deterioration of mood. Eating disorders, self-blame, or suicidal ideation are not uncommon [2]. It is assumed that depressive disorders in patients with chronic cerebrovascular diseases are associated with specific neurochemical disorders caused by damage to frontal and frontotemporal structures.

At the same time, most patients with pre-dementia forms of NCF develop anxiety disorders, which can be seen not as a consequence of organic brain damage, but as a reaction to disturbances in mental activity, fear of illness. development, working capacity, concerns about social functions, etc. It should also be taken into account that many patients cannot describe their condition, as a result of which they complain of various symptoms: cephalalgia, myalgia, arthralgia, pain in internal organs and other unpleasant sensations, the occurrence of which cannot be explained by somatic or neurological pathologies. Often, dyssomnia disorders (difficulty falling asleep, early morning awakenings) and emotional lability (increased excitability, tearfulness, touchiness) are observed simultaneously [8, 9]. As the disease progresses, anxiety and depressive disorders are pushed back, and indifference to oneself and others, decreased motivation, and lack of initiative appear and intensify [8].

In addition to NCF and affective disorders, patients with chronic cerebrovascular diseases may experience motor disorders (extrapyramidal syndrome, elements of central paresis, vestibulo-atactic disorders), pseudobulbar syndrome, and less often sensory disorders. The severity of clinical manifestations, as well as the characteristics of the combination of signs of damage to different parts of the brain, determined by the nature, location and degree of damage to the brain matter, may vary.

Currently, a lot of information is presented about the medical and social importance of the problem of dementia, in particular, about the high number of patients with NCF, the high probability of further progression and spread of the disease, the large financial costs associated with treatment and life support of patients [16]. At the same time, some reports contain a more optimistic assessment of the problem and allow us to consider dementia (Alzheimer's type and vascular and mixed) as a potentially preventable condition.

A number of large European studies (Sweden, the Netherlands, the UK, Spain) have been devoted to studying the dynamics of the development of various types of dementia in recent decades [21]. According to their results, a decrease in the prevalence of dementia was noted in some countries (for example, in the UK by 22%), while in Spain the dynamics were noted in men (a decrease in prevalence by 43%) and to a lesser extent among women. Analyzing the data obtained, the authors concluded that people who lead a healthy lifestyle in early and middle age can reduce the likelihood of dementia in late life.

In general, similar results were obtained in a 10-year prospective study conducted in the United States, the aim of which was to examine the association of ideal cardiovascular health status (a set of criteria proposed by the American Heart Association) with the risk of developing cerebrovascular diseases and dementia [18]. Compliance with the established criteria (smoking cessation, regular physical activity, rational nutrition, maintaining optimal BMI, blood pressure, cholesterol and glucose levels in the blood) can reduce not only stroke, but also vascular dementia. Mixed types of Alzheimer's and dementia, as well as brain atrophy. According to the authors, compliance with these criteria from a young age is of fundamental importance.

Similar data were obtained to some extent in the United States from 1982 to 1999. A decrease in the incidence of severe dementia was observed among patients registered in the Medicare health system [17]. However, despite the decrease in the prevalence of severe forms of dementia in the United States,

the authors are very cautious about the sustainability of this trend. In their opinion, the number of patients with cognitive impairment may increase further, which is associated with an increase in the average age of the population, associated diseases and other reasons. Further study of factors that reduce the incidence of dementia and methods of slowing the progression of the disease can serve as the basis for evidence-based prevention strategies.

In clinical practice, it is not always possible to implement the principles of disease prevention, in particular, the prevention of cognitive decline, and therefore doctors of various specialties (neurologists, family doctors, psychiatrists, etc.) often have to deal with patients. drug therapy is needed to correct already developed NKFs. Currently, acetylcholinesterase inhibitors and modulators of glutamate release from the presynaptic cleft are the most common for the treatment of patients with dementia. The high effectiveness of these drugs has been shown in double-blind, multicenter, randomized clinical trials. At the same time, nootropic drugs, drugs with neuroprotective and neuroreparative properties, herbal preparations, and many other substances are often prescribed. The effectiveness of these drugs has not always been proven in well-planned and strictly organized clinical studies, but significant experience in their practical use shows that they can achieve a significant clinical effect in the treatment of many patients.

In general, drugs that affect various metabolic processes in the body and in the brain in particular are of undoubted interest. These include Cytoflavin, which is available in two forms: ampoules (active ingredients in a 10 ml ampoule: succinic acid - 1 g, nicotinamide - 0.1 g, riboxin - 0.2 g, riboflavin - 0.02 g) and tablets (active ingredients in one tablet: succinic acid - 0.3 g, riboxin - 0.05 g, nicotinamide - 0.025 g, riboflavin - 0.005 g). According to modern ideas about the role of succinic acid preparations in the processes of metabolism and energy metabolism in the brain, the pharmacological effect of Cytoflavin is associated with the complex effect of its components. Cytoflavin helps activate aerobic metabolism in neurons and glial cells, which increases the intensity of glucose utilization, helps activate β-oxidation of fatty acids, and replenishes the deficiency of γ-aminobutyric acid in neurons (which is important, since it is the main inhibitory neurotransmitter in the central nervous system).

Cytoflavin increases the resistance of nerve and glial cell membranes to ischemia. This phenomenon is manifested by a decrease in the concentration of neurospecific proteins in the blood, which characterize the degree of destruction of the main components of nervous tissue [1].

Cytoflavin improves cerebral blood flow, activates metabolic processes in the central nervous system, and also restores impaired consciousness if its depression is not associated with systemic damage to the activating reticular formation (for example, during poisoning with neurotropic poisons that depress brain activity, during poisoning with alcohol and drugs for inhalation anesthesia) [1]. Cytoflavin contributes to the regression of neurological symptoms and the improvement of cognitive functions of the brain, regardless of the nature of brain damage.

A number of studies have investigated the use of Cytoflavin in patients with acute ischemic stroke [1, 11]. According to the results of a number of preliminary studies with an open design, the use of Cytoflavin in patients with acute ischemic stroke has a positive effect: complete restoration of motor functions of paralyzed limbs and a reduction in treatment time. At the same time, good tolerability of the drug and the absence of significant side effects during its use were noted.

The positive results of the initial studies served as the basis for conducting an open randomized clinical and instrumental pilot study after registration, which studied the efficacy and tolerability of a stepwise regimen of Cytoflavin as part of the main therapy in patients with acute ischemic stroke. middle cerebral artery [13]. The experiment included clinical observation and dynamic neuroimaging (MRI). The study was conducted in Russia in 2008-2009. A total of 70 patients were included on the basis of 5 clinical centers.

According to the approved protocol, patients with rapid regression of clinical manifestations, intracranial hemorrhage, cancer, drug addiction and somatic diseases in the stage of decompensation were not included in the study. The use of other antioxidant or nootropic drugs during the study period was excluded. Patients in the main group (n = 41, 22 men and 19 women; mean age - 64.4 ± 9.0 years)

received Cytoflavin according to the following scheme from the 1st day of the disease: 1-10 days - intravenous drip 20 ml. 400 ml of 5% glucose solution or 0.9% sodium chloride solution 2 times a day; 11-35 days - orally, 425 mg 2 times a day, half an hour before meals, without chewing, with 100 ml of water, no later than 18:00, comparable to patients in the comparison group. The main group, according to clinical and demographic indicators, did not receive Cytoflavin.

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