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Clinic, Diagnostics and Treatment of Cerebral Arterial Insufficiency

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Abstract

Currently, the problem of cerebrovascular diseases is extremely relevant for a neurologist. Patients with stroke and chronic cerebral vascular insufficiency also regularly come to the attention of cardiologists, therapists and doctors of other specialties. Chronic cerebrovascular insufficiency is one of the main causes of cognitive impairment and dementia, as well as disability in old age.

Key words: Diagnostics, Clinic, Arterial, *Patients*

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Introduction

In domestic neurological practice, the syndrome of chronic vascular progressive brain damage is designated by various terms, such as "dyscirculatory encephalopathy", "chronic cerebral ischemia", "ischemic brain disease" and some others. From our point of view, the main pathogenetic mechanisms of chronic progressive brain damage are most fully reflected in the concept of dyscirculatory encephalopathy (DE). Academician of the Russian Academy of Medical Sciences, prof. N.N. Yakhno proposed to understand the term DE as a combined brain damage as a result of repeated acute cerebrovascular accidents and chronic cerebral blood supply insufficiency [2, 3]. In this case, acute cerebrovascular accidents can occur both with and without stroke clinical symptoms, such as "silent" infarctions and/or hemorrhages. Etiology and pathogenesisThe most common causes of DE are cerebral atherosclerosis, arterial hypertension, cardiovascular diseases with a high risk of embolism to the brain (for example, atrial fibrillation, heart valve pathology, ischemic heart disease, etc.) (Fig. 1). In this case, cerebral arterial insufficiency is usually combined with other systemic signs of underlying vascular disease. In particular, cerebral vascular atherosclerosis is often combined with ischemic heart disease and peripheral arterial insufficiency. Therefore, the presence of these pathological conditions should increase medical alertness regarding the increased risk of cerebral vascular disease. The treatment program for patients with DE syndrome (or a history of strokes) should, if possible, include drugs that have a systemic effect on peripheral blood flow [2, 3, 7, 19].

Due to some anatomical and physiological features of cerebral circulation, there are parts of the brain that are more or less "vulnerable" to ischemic damage. Physiologically, the deep structures are in the most "unfavorable" position: the subcortical gray nodes and the periventricular white matter of the cerebral hemispheres. According to statistics, it is here that focal and diffuse changes in the brain matter associated with ischemic damage are formed earlier and most often. At the same time, even the so-called "silent infarctions" are not asymptomatic, but are manifested by cognitive and emotional disorders that are not associated in the minds of doctors with a stroke and therefore are unrecognized [4, 5, 8, 22]. The deep parts of the cerebral white matter are located on the border of the carotid and vertebrobasilar basins ("watershed zone"), therefore they suffer from damage to the main arteries of the head, for example, as a result of atherosclerosis. Microangiopathy of penetrating arteries due to long-standing uncontrolled arterial hypertension, diabetes mellitus or other diseases affecting small-caliber vessels also leads to damage of the above-mentioned areas. Thus, damage to both large and small vessels can lead to damage of subcortical structures and deep areas of the white matter of the brain. As a result of pathology of the deep areas of the white matter, a “disconnection phenomenon” is formed: a disruption of the connection between the cortical and subcortical areas of the brain, which causes the main clinical manifestations of DE. They are primarily based on dysfunction of the frontal lobes of the brain. This is due to the special psychophysiological role of the frontal lobes, which plan and control cognitive activity and voluntary behavior. Disturbances in communication with other cerebral structures significantly complicate the implementation of this function [2, 5, 7, 19]. Clinical picture Despite the highly variable clinical picture of ED, in the overwhelming majority of cases, emotional and cognitive symptoms of dysfunction of the frontal lobes of the brain are present at the early stages, which reflects the pathogenetic basis and localization of the pathological process in ED. These symptoms can serve as "indicative" for the early diagnosis of chronic vascular damage to the brain. In the early stages of ED, cognitive disorders are determined in approximately 90% of patients [11-13]. At the stage of mild cognitive impairment, the rate of thought processes and concentration decrease, episodic forgetfulness and increased fatigue during mental stress occur. A detailed neuropsychological

examination is necessary to diagnose mild cognitive disorders. Simple screening methods, such as the Mini-Mental State Examination, the Clock Drawing Test, and the Frontal Function Battery, usually do not reveal any impairments [3, 10, 12, 13]. Moderate cognitive impairment is indicated by its more persistent and definite nature. In this case, memory impairment and other cognitive impairments clearly go beyond the age norm, but do not reach the severity of dementia [11, 12, 24]. The most typical impairments are impairments in planning and control of activities, intellectual processes, the ability to generalize and draw conclusions, which also reflects dysfunction of the frontal lobes of the brain. In typical cases, memory impairment is mild, such as difficulty reproducing information while the ability to memorize is preserved. Memory of life events remains mostly intact. In the absence of strokes, speech and information perception (gnosis) impairments are not typical [7–12].

Vascular dementia is the most extreme manifestation of vascular cognitive impairment and usually develops many years after the onset of the pathological process. The development of vascular dementia indicates the onset of stage III of dementia. Evidence of the transformation of moderate cognitive impairment into dementia is the formation of a patient's dependence on outside help due to cognitive insufficiency. The presence of such dependence is indicated, in particular, by the impossibility or significant difficulties in independent interaction with a doctor, when the patient cannot accurately tell the history of the disease, does not follow the doctor's recommendations due to forgetfulness or difficulties in organizing activities [13, 21]. As mentioned above, before the formation of vascular dementia, and often at the stage of mild dementia, the presence of cognitive impairment may not be obvious during the routine collection of complaints and anamnesis. To objectify the cognitive status when working with elderly patients with atherosclerosis and other vascular diseases, neuropsychological methods should be used. The Mini-Cog test is used as the simplest screening method, which is a test for memorizing three words, where the patient is asked to draw a clock with numbers and hands as an interfering task [20]. However, it should be noted that this method is uninformative at the stage of non-dementia cognitive disorders. For a more accurate assessment of cognitive status, the Montreal cognitive scale is currently being actively positioned, which contains tests for frontal functions (test of the connection of numbers and letters, choice reaction, forward and backward counting), memory, orientation, drawing geometric figures, etc. (Fig. 2). Emotional disorders in dementia Very often, vascular cognitive disorders are combined with emotional disorders in the form of vascular depression, emotional lability, decreased motivation and apathy. Depression in dementia is usually organic in nature due to the dissociation of brain structures and the development of secondary dysfunction of the frontal lobes. According to studies conducted in the clinic of nervous diseases, depression is noted in approximately 70% of dementia cases. At the same time, patients themselves rarely complain of depression or a decrease in mood. The most characteristic symptom is a painful fixation on unpleasant somatic sensations that cannot be fully explained by existing diseases. Typical complaints include headaches, back pain, joint pain, internal organ pain, dizziness, noise and ringing in the head. Vascular depression is characterized by a protracted course and poorly responds to antidepressant therapy [10, 19, 21]. Another characteristic type of vascular emotional disorders is emotional lability, which is a rapid change in mood, a tendency to explosive reactions. Episodes of uncontrollable crying are noted, which occurs for little reason, irritability and aggression towards others [10, 19, 21]. Vascular depression and emotional lability are usually noted at earlier stages of DE and are combined with mild or moderate cognitive impairment. At later stages of this syndrome, and especially in patients with established vascular dementia syndrome, a more common emotional disorder is apathy. It is manifested by decreased motivation and independent urges for any activity. Patients lose interest in their previous hobbies, do nothing most of the time or engage in unproductive activities [8, 10].

Diagnosis of VE To diagnose VE syndrome, a thorough study of the disease history, assessment of the neurological status, and the use of neuropsychological and instrumental research methods (primarily neuroimaging) are necessary. It is important to emphasize that the presence of cardiovascular diseases in an elderly person in itself is not yet proof of the vascular nature of the detected neurological disorders. A necessary condition for correct diagnosis is obtaining convincing evidence of a cause-and-effect relationship between neurological and cognitive symptoms and cerebrovascular pathology (Table 1). Neuroimaging methods play an important role in examining patients and establishing the vascular nature of symptoms: computed X-ray or, preferably, magnetic resonance imaging of the brain. This research method allows visualizing the consequences of acute cerebrovascular accidents and diffuse changes in the white matter (leukoaraiosis). The presence of these changes is indisputable evidence of vascular damage to the brain [6, 13, 18]. Treatment Treatment of cerebral circulatory insufficiency should be aimed at both the underlying pathological processes of cerebrovascular insufficiency, such as arterial hypertension, atherosclerosis of the main arteries of the head, heart disease, and the pathogenetic mechanisms of DE: cerebral ischemia and hypoxia [2, 3, 16]. Pathogenetically, it is justified to prescribe drugs that improve cerebral microcirculation, but do not cause the steal effect. Such drugs include: phosphodiesterase inhibitors: pentoxifylline, vinpocetine, ginkgo biloba preparations, etc. The vasodilatory effect of these drugs is associated with an increase in the content of cAMP in the smooth muscle cells of the vascular wall, which leads to their relaxation and an increase in the lumen of the vessels; calcium channel blockers: cinnarizine, flunarizine, nimodipine. They have a vasodilating effect by reducing the intracellular calcium content in the smooth muscle cells of the vascular wall; α2-adrenergic receptor blockers: nicergoline, piribedil. These drugs eliminate the vasoconstrictive effect of sympathetic nervous system mediators: adrenaline and noradrenaline, and also, due to the effect on presynaptic receptors, increase the activity of noradrenergic mediation in the brain. The latter has an additional nootropic effect, most noticeable in relation to memory and regulation of voluntary activity. One of the most highly effective vasoactive drugs for the treatment of cerebral and peripheral arterial insufficiency is pentoxifylline (Trental). Trental is a derivative of dimethylxanthine. The advantage of this drug is its systemic effect on microcirculation: the drug affects both cerebral and peripheral vessels without causing a steal effect. The mechanism of action of this drug is associated with the inhibition of phosphodiesterase type 4, which leads to an increase in the content of cyclic AMP in the smooth muscle cells of the vascular wall and blood cells. The accumulation of cyclic AMP in platelets and erythrocytes has some antiaggregant effect, contributes to an increase in the deformability of blood cells and a decrease in its viscosity. In occlusive lesions of peripheral arteries (for example, with intermittent claudication), the effect of the drug is manifested in an increase in walking distance, elimination of night cramps in the calf muscles and the disappearance of pain at rest [14, 25, 26].

The use of Trental in chronic cerebrovascular insufficiency and peripheral circulatory disorders is based on both the results of controlled randomized studies and many years of practical experience. In 2003, M. Sha et al. conducted a meta-analysis of 20 randomized controlled double-blind studies in patients with vascular cognitive disorders, performed in European countries. It was shown that against the background of the use of Trental in patients with vascular dementia and less severe cognitive impairment, statistically significant positive dynamics of cognitive indicators and other neurological disorders are noted [26]. In order to clarify the effectiveness of pentoxifylline in patients with chronic cerebrovascular disorders, an examination of 55 patients with the consequences of mild stroke was conducted in the outpatient and polyclinic network of Moscow, who received a prolonged form of Trental 400 mg at a daily dose of 1200 mg / day. During therapy, a significant improvement in memory, attention, and other cognitive functions was noted. The most pronounced effect of pentoxifylline in chronic cerebrovascular disorders, consisting

in slowing the progression of cognitive impairment, was observed in patients with multi-infarct dementia, but not with other types [1]. Another study of the effectiveness of pentoxifylline in 289 patients with multi-infarct dementia was conducted in a European double-blind, placebo-controlled, parallel multicenter study. For 9 months, treatment was carried out with this drug at a daily dose of 1,200 mg or placebo. During therapy, a significant improvement in cognitive functions in patients was noted [14]. The high effectiveness of Trental in vascular pathology of the brain was reproduced in an open multicenter study involving 10,423 patients with various cerebrovascular diseases. The drug was used at a dose of 300 to 600 mg / day. for 8 weeks, which led to a reliable improvement in memory, mood and other symptoms. Regression of symptoms such as tinnitus, headache, dizziness, was noted in 76-87% of cases. It is also important to note the good tolerability of Trental. The incidence of side effects with this therapy was less than 0.25% [17]. S. Takamatsu et al. reported an improvement in the clinical condition of patients with chronic cerebrovascular disorders when they were prescribed Trental at a dose of 300 mg / day. This improvement was combined with a reliable decrease in the levels of fibrinogen and a1-antitrypsin [26]. L. Parnetti et al. used Trental at a dose of 1200 mg / day in 30 patients with cognitive disorders; 30 similar patients received placebo. Therapy was carried out in two courses of 12 weeks with a break of 4 weeks between them. When compared with placebo, the drug was shown to be able not only to improve cognitive abilities according to psychometric methods, but also to have a beneficial effect on the rheological properties of the blood. Thus, there was a decrease in blood viscosity and an increase in the filterability of erythrocytes. The improvement was most pronounced after the second period of treatment. According to the authors, this proves that long-term therapy is necessary to obtain optimal results [23].

Summarizing the available data, we can say that the best therapeutic efficacy of Trental is observed when using a daily dosage of 1200 mg. In domestic practice, vasoactive drugs are usually prescribed in courses of 2-3 months, 1-2 times a year. However, in recent years, the advisability of longer vascular therapy has been widely discussed. Metabolic therapy is widely used for cerebrovascular insufficiency. The purpose of this type of treatment is to stimulate the reparative processes of the brain associated with the phenomenon of neuronal plasticity. In addition, metabolic drugs have a symptomatic nootropic effect. Neurometabolic drugs include piracetam, actovegin, cerebrolysin, choline alfoscerate, etc. [2, 10]. Like treatment with vasoactive drugs, metabolic therapy is administered in courses 1–2 times a year. Combined vasoactive and metabolic therapy is pathogenetically justified and appropriate. The development of dementia syndrome requires additional administration of drugs that primarily affect synaptic transmission processes. It has been established that acetylcholinesterase inhibitors (donepizil, rivastigmine, galantamine, ipidacrine) and the NMDA receptor blocker memantine have the greatest clinical efficacy in dementias of various etiologies. Against the background of the use of these drugs, a regression in the severity of cognitive disorders is observed, the behavior of patients improves, they become less dependent on outside help. Treatment with acetylcholinergic or glutamatergic drugs should be carried out for a long time, possibly permanently [2, 16, 28]. In conclusion, it should be emphasized that a comprehensive assessment of the state of the cardiovascular system of patients with cerebrovascular insufficiency, the impact on both the cause of the disorders and the main symptoms of DE contribute to an improvement in the quality of life of patients and their relatives.

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