

Measures for the Diagnosis and Treatment of Chronic Cerebral Ischemia

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Abstract: Clinical manifestations of HIU are considered depending on the severity of the pathological process. Special attention is paid to vascular cognitive impairment in various lesions. An original quantitative chemical scale adapted for outpatient practice is proposed. The treatment of patients with GIU is discussed based on the pathogenesis of the disease. Methods of hypotensive and lipid-lowering therapy, cerebrovascular, antiplatelet, the appointment of neurometabolic drugs and antioxidants is considered.

Keywords: cerebrovascular disease, chronic cranial ischemia, pathogenesis and treatment.

Cerebrovascular diseases (CVD) are one of the main causes of morbidity and mortality. Epidemiology of chronic forms of TsVK has not been fully studied until now. When the first symptoms of cerebrovascular insufficiency appear, the patient is usually referred to a local therapist (general practitioner, family doctor) and only with specific symptoms - to a neurologist. The development of a unified approach and algorithm for the diagnosis and treatment of the initial forms of chronic TsVK by these specialists mainly determines the rate of development of the disease, prevention of stroke and dementia.

Chronic cerebral ischemia (CMI) is a disease caused by progressive multifocal diffuse brain damage, manifested by various degrees of neurological disease due to decreased cerebral blood flow, repeated transient ischemic attacks or cerebral infarctions.

In terms of the etiology and pathogenesis of SMI, the boundary between acute and chronic forms is very conditional in terms of molecular mechanisms of neuronal damage. Atherosclerosis, hypertension, myocardial diseases, diabetes and other diseases play a leading role in the etiology of SMI. All of them lead to changes in cerebral blood flow with hypoxia and a cascade of biochemical changes, which lead to diffuse multifocal changes in the brain substance. Due to a decrease in cerebral blood flow, the main factors underlying the pathogenesis of SMI are the disruption of cerebral microhemocirculation, insufficient energy resources to ensure the functioning of the brain, and glutamate excitotoxicosis. Excessive activation of the glutamatergic system leads to a large uptake of calcium and sodium ions into neurons, depolarization of cell membranes, activation of voltage-dependent calcium channels and intracellular accumulation of calcium, resulting in a cascade of negative pathobiochemical processes with lactic acidosis, activation of intracellular enzymes, nitric oxide and calcium synthesis increases. development of oxidative stress. Disturbance of the rheological properties of blood and brain microhemocirculation is of great importance. It is known that blood flow in capillaries depends on the elasticity and deformation of erythrocytes, whose diameter is several times larger than the diameter of capillaries. In atherosclerosis, due to the accumulation of cholesterol in the membranes of erythrocytes, the deformation of cells changes, spheroid and folded forms and conglomerates of erythrocytes appear in the blood, the aggregation of erythrocytes and platelets increases, the rheological properties of blood change and its viscosity increases.

Ischemic "penumbra" (penumbra) zones appear in the brain as a result of impaired cerebral circulation. This is because the critical blood flow for cellular electrical activity is 20 ml/100 g per minute, and the critical blood flow for cellular pumps and maintenance of cellular homeostasis is 15 ml/100 g per minute. Cells between these two critical levels form an ischemic "penumbra" in which cellular functions can be restored. Decreased blood flow and ischemic "penumbra" zones have a mosaic

character. The distribution of the ischemic "penumbra" depends on the condition of the collateral circulation in the adjacent circulatory areas and the rheological properties of the blood.

102 patients participated in the study, and all patients were examined. Clinical presentation The diagnostic algorithm of SMI consists of complaints and anamnesis, somatic and neurological examination, if necessary, measurement of blood pressure (AB) in two arms - daily monitoring, electrocardiogram registration, Holter monitoring, ultrasound Doppler and duplex scanning of the precerebral arteries in the neck, transcranial (intracranial) dopplerography, laboratory research methods with lipidogram, including total cholesterol, triglycerides, high, low and very low density lipoproteins and calculation of total cholesterol. atherogenic index. In cases of unclear diagnosis, neuromagnetic (computed X-ray or magnetic resonance imaging of the brain) is performed.

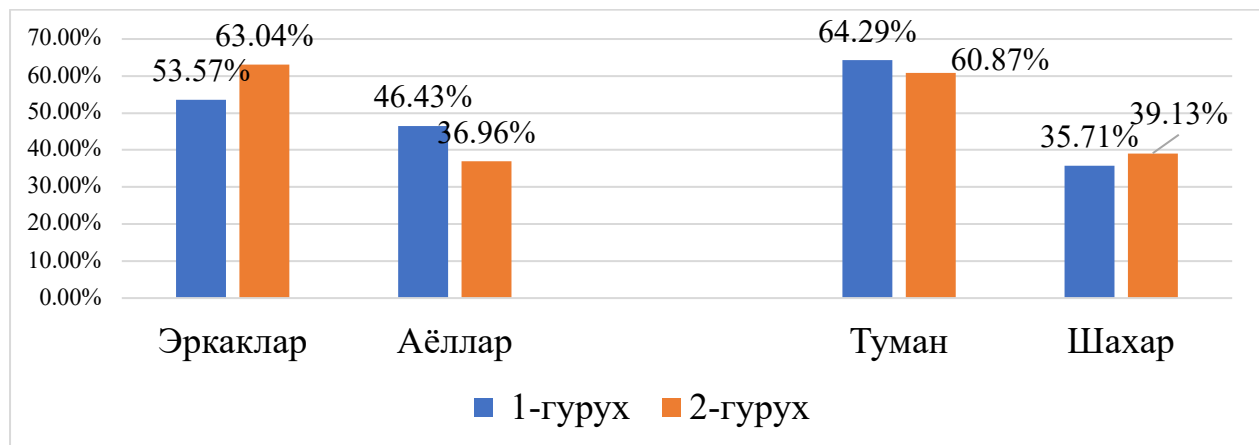


Figure -1. Age and gender composition of the main group of patients ($p>0.05$)

The Memory Impairment Screening (MIS) was performed through a primary memory assessment. In the main and comparative group

Clinical manifestations of SIM depend on the severity and spread of the process, general brain symptoms (headache, dizziness), asthenic syndrome, anxiety-depressive disorders, cognitive impairment and focal neurological symptoms can be distinguished in various combinations. Most of these syndromes are manifested in the complaints of patients, and the test questions skillfully asked by the doctor show the nature of the disease.

Vascular diseases in the anamnesis: mostly elderly and old age, arterial hypertension (AG), coronary artery disease, atherosclerosis of peripheral arteries. Headaches are associated with overwork after physical and emotional stress and decrease after a short rest. If the patient has arterial encephalopathy combined with venous, then the nature of the headache will be different. Patients complain of morning or night headaches, dizziness, changes in body position, noise in the head or ears, worsening of symptoms when wearing tight collars or straps, sleeping on a low pillow, discomfort, eye fatigue. in the morning ("sand in the eye" symptom), morning swelling on the lower surface of the eye and eyelids (pale, purple cyanotic color) or mild nasal congestion (outside of the symptoms of an acute respiratory viral infection). Many patients have asthenic complaints (increased fatigue, decreased performance, poor sleep). Excessive anxiety, insecurity, bad mood, excessive anger and complacency appear. Somatized depression is manifested in many complaints of dysfunction of internal organs. Patients note deterioration of memory for current events, forgetfulness in everyday situations. Intellectual characteristics, social adjustment and daily activities are not impaired.

Complaints may be associated with focal neurological symptoms. It is necessary to determine if there is a decrease in vision in one eye, which may be a sign of stenosis of the internal carotid artery in the neck with a blood circulation disorder in the orbital artery; noise or ringing in the ears, hearing loss, tremors when walking, gait changes.

Vascular cognitive disorders include the most complex functions of the brain, such as memory, speech, intelligence, purposeful motor activity (praxis) and holistic perception. The term "vascular cognitive

impairment" (VCD) was introduced to denote impairment of higher brain functions due to cerebrovascular pathology. Cognitive impairment is most common in stroke patients with chem.

Patients with SMI stage i with a mild form of vascular cognitive disorders are characterized by complaints of memory loss, performance impairment, and inattention. Memory loss primarily concerns current events, surnames, names and telephone numbers. Patients are increasingly forced to use notebooks. Changes in the cognitive field are not visible to others, but are felt by the patient himself and are confirmed by careful examination using neuropsychological methods.

Patients with moderate CKD complain of increased weakness or decreased mental ability. The clinical presentation is dominated by memory impairment for current events that is slowly progressive in nature. In another variant, several cognitive functions are impaired: memory, spatial orientation, intelligence and praxis. But cognitive impairment does not lead to the loss of professional skills or social interaction skills, although there may be some deterioration in complex and instrumental types of daily and professional activities.

Dementia is a severe form of SMI. According to the definition of the World Health Organization, dementia is an acquired global disorder of all higher cortical functions, including memory, the ability to solve problems of everyday life, to perform acquired complex actions, the correct use of social skills, all aspects of language. and control of emotional reactions in the absence of communication. loss of consciousness. In the stage of dementia, the patient needs complete external care. This is the most severe clinical syndrome of cognitive impairment.

Dementia develops in the later stages of the development of SMI and can occur as a result of the development of lesions in certain areas of the brain and the disruption of interneuronal relationships with damage to deep parts of the brain.

Principles of therapy. Treatment of patients with SMI based on the pathogenesis of the disease includes hypotensive and hypolipidemic therapy (according to the instructions), cerebrovascular, antiaggregant, neurometabolic and neuroprotective drugs, antioxidants. Not all hypertensive drugs are suitable for the treatment of AG and SMI

In patients with disorders in the system of autoregulation and reactivity of cerebral arteries, a rapid and uncontrolled hypotensive effect can lead to hypoperfusion of the brain. When prescribing antihypertensive drugs, adverse chemical reactions should be taken into account, and hypertensive therapy should be started with particular caution. These include critical stenosis on ultrasound duplex scanning of additional cranial and intracranial arteries or combined and multiple stenoses of the main arteries of the head, stage II-III SMI in patients diagnosed with stenosis of intracranial arteries. In such patients, at the beginning of treatment, the decrease in systolic blood pressure AB(SAB) should not exceed 15%, diastolic - 10% of the initial level. Elderly and elderly patients should be based on combined therapy in the form of a combination of angiotensin-converting enzyme inhibitor and calcium antagonist. Calcium channel blockers (dihydropyridines) - amlodipine - improve cerebral blood flow by dilating arterioles and improving endothelial function. Therefore, they are considered first-line drugs in the treatment of hypertensive patients with TsVK.

Angiotensin-converting enzyme inhibitors have the ability to dilate small and medium-sized arteries, in particular, the branches of the middle cerebral artery, as a result of which cerebral blood flow remains sufficient even when blood pressure is below the minimum threshold of autoregulation.

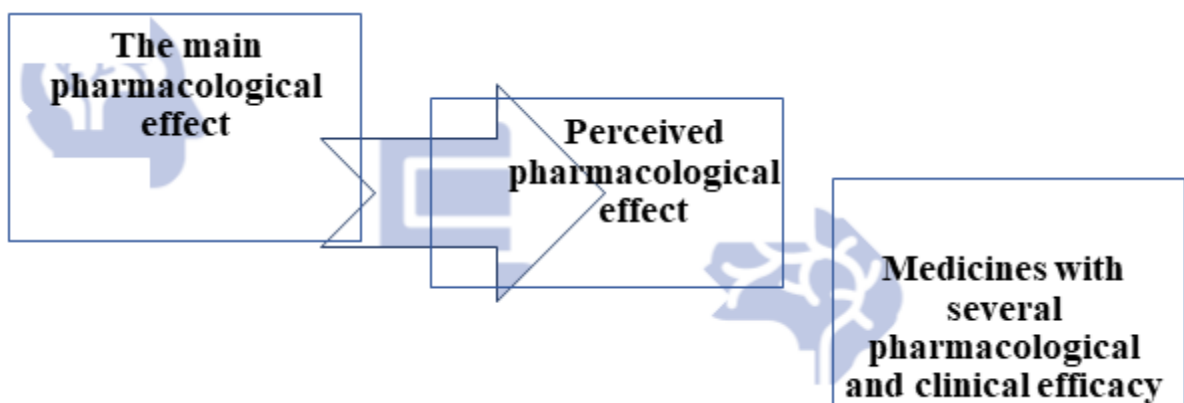
It is known that a serious problem is the low adherence of patients to antihypertensive therapy, the reasons for which are diverse. Patients are affected by cognitive disorders: memory loss (they forget to take medication), forgetfulness and carelessness (irregular medication intake) and decreased criticality of their condition. Refusal to take antihypertensive drugs may also be due to the fact that these drugs do not relieve many neurological symptoms (dizziness, headache, unsteadiness while walking, increased anxiety, irritability) typical for SMI.

One of the options for a therapeutic strategy that increases patients' adherence to antihypertensive therapy may be the simultaneous use (in combination with antihypertensive drugs) of drugs that improve cognitive and vestibular functions and reduce headache. The most appropriate task (in terms of the variety of clinical and pharmacological effects and the width of the therapeutic spectrum) is the group of nootropic drugs. The main mechanisms of the effect of nootropic (neurometabolic) therapy are influencing the energy exchange in the brain, increasing the resistance of the brain to hypoxia and ischemia, facilitating learning and memory processes, improving concentration and information processes in the brain. These drugs should be prescribed differentially depending on the severity of cognitive impairment. Comprehensive chemical treatment should include measures to reduce the effects of lipid peroxidation products, strengthen the physiological antioxidant system, and prevent the formation of reactive oxygen species and other free radicals. In recent decades, the anti-ischemic activity of succinic acid, its salts and esters, which are universal intracellular metabolites, has been studied. Succinic acid, present in organs and tissues, is a product of reaction 5 and a substrate of reaction 6 of the tricarboxylic acid cycle. In the 6th reaction of the Krebs cycle, the oxidation of succinic acid is carried out using succinate dehydrogenase. Performing a catalytic function in relation to the Krebs cycle, succinic acid reduces the concentration of lactate, pyruvate and citrate, produced in the initial stages of hypoxia, in the blood of other intermediate substances of this cycle.

The antihypoxic effect of succinic acid is related to its effect on the transport of intermediary amino acids, as well as an increase in the content of γ -aminobutyric acid in the brain. The ischemic effect of succinic acid is associated not only with the activation of succinate dehydrogenase oxidation, but also with the restoration of the activity of cytochrome oxidase, the main oxidation-reduction enzyme of the respiratory mitochondrial chain.

We divided the patients into two groups. Group 1 patients were given vitamin V1, V6, V12 complex (milgamma) intramuscularly 1 time per day for 10 days, then 1 tablet 2 times a day for 2 months, folic acid 1 tablet 3 times a day orally for 1 month.

The 2nd group of patients (the main group) was given ethylmethylhydroxypyridinesuccinate as an antioxidant and antihypoxant for 10 days with the addition of 10 ml of sodium chloride 0.9%-100 ml, then 125 mg of 1 tablet 3 times a day for 3 months. Galantamine and ethylmethylhydroxypyridinesuccinate were given in addition to the main group drug to improve cognitive function. Vitamin V1, V6, V12 complex (milgamma) as the main group of drugs was administered intramuscularly 1 time per day for 10 days, then 1 tablet 2 times a day for 2 months, folic acid in the form of 1 tablet 3 times a day orally for 1 month.



Picture-2. Pathogenetic and several related pharmacological and clinical implications

As mentioned, the desired main effects of multimodal pharmacotherapy of SMI include influencing the energy metabolism of the brain, improving cerebral blood circulation in the macro- and microcirculation system. From the point of view of multimodal pharmacotherapy, the appointment of piracetam can be considered and has an advantage, its main effect is the improvement of energy-dependent processes of the brain and effects in the form of vasoactive effects - a decrease in cerebral

blood vessels, a decrease in flow and peripheral vascular resistance with an increase in volumetric cerebral blood, and a decrease in platelet aggregation and effect on the rheological properties of blood with increased deformation of erythrocytes. But at the same time, it is necessary to take into account the dose-dependent effect of piracetam, as a result, it is advisable to use high-dose tablet drugs. It is clear that it is unreasonable to prescribe cerebrovascular drugs and antiplatelet agents when prescribing piracetam.

When prescribing antioxidants, their associated cognitive effects should be taken into account. Mexidol (succinic acid) and cytoflavin (succinic acid, nicotinamide, riboxin, riboflavin) have antioxidant properties. A judicious combination of monomodal and multimodal pharmacotherapy helps avoid polypharmacy and more effectively treat patients with SMI.

In conclusion, it should be noted that timely diagnosis and treatment provided positive results for the patients in the study.

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