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ALGORITHM FOR THE DIAGNOSIS AND TREATMENT OF DISCIRCULATORY ENCEPHALOPATHY IN PATIENTS WITH ARTERIAL HYPERTENSION

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Abstract
In clinical practice, both overdiagnosis and underdiagnosis of DE are often observed. To make a correct diagnosis, it is necessary, along with obtaining detailed medical history data, a thorough examination of the neurological status and the state of internal organs (primarily the state of the cardiovascular system), to conduct a number of paraclinical research methods, among which the leading ones are neuroimaging, neuropsychological methods and methods of studying blood vessels supplying the brain.

Keywords: dyscirculatory encephalopathy, algorithm, arterial hypertension, diagnostics, cerebrovascular disorders.

The term "dyscirculatory encephalopathy" (DE) refers to a chronic cerebrovascular pathology that develops with multiple focal and/or diffuse brain lesions [1, 2]. At the same time, the progression of neurological and mental disorders may be due to persistent and prolonged cerebrovascular insufficiency and / or repeated episodes of dyscirculation, occurring both with acute clinical symptoms (acute cerebrovascular accidents) and subclinically. The term "dyscirculatory encephalopathy" was proposed by G.A. Maksudov and V.M. Kogan in 1958. This term is not mentioned in ICD-9 and ICD-10, and among conditions similar in clinical presentation are: cerebral atherosclerosis, progressive vascular leukoencephalopathy, hypertensive encephalopathy, other unspecified lesions of cerebral vessels, including cerebral ischemia (chronic) and cerebrovascular disease, unspecified. Some authors also suggested other names: ischemic brain disease [3], vascular encephalopathy, or angioencephalopathy [4]. However, the term "dyscirculatory encephalopathy" turned out to be more competitive and preferable due to the fact that it denotes not only the morphological basis of the disease, but also its pathogenesis. DE is heterogeneous, which is reflected in the etiology, clinical, neuroimaging and morphological features of its individual forms. The following main variants of DE can be distinguished [5]:

1. Hypertensive DE. 1.1. Subcortical arteriosclerotic encephalopathy (SAE). 1.2. Hypertensive multi-infarct encephalopathy.
2. Atherosclerotic DE.
3. Chronic vascular vertebrobasilar insufficiency.
4. Mixed shapes.

Binswanger's disease (subcortical arteriosclerotic encephalopathy)

Arterial hypertension (AH) is the main etiological factor in the development of SAE. The morphological picture of the SAE is presented [4, 6, 7]:

• areas of diffuse lesions of the white matter (mainly periventricular) with many foci of incomplete necrosis, loss of myelin and partial collapse of the axial cylinders, foci of encephalolysis, diffuse proliferation of astrocytes;
• diffuse spongiosis, more pronounced periventricular;
• lacunar infarcts in the white matter, subcortical nodes, thalamus, pons, cerebellum;
• thickening and hyalinosis of small arteries (arteriosclerosis) in the white matter and gray matter of the basal ganglia; • hydrocephalus due to a decrease in the volume of white matter.

The pathology of the white matter in SAE is based on arteriosclerosis of arterioles and small arteries (>150 μm in diameter). The cause of diffuse ischemia of the white matter and the development of focal lesions (lacunary infarcts) is a decrease in perfusion due to widespread stenosis and occlusion of small cerebral arteries [8].

Neuroimaging studies of the brain in patients with SAE are observed [4, 9]:

• leukoaraiosis - a decrease in the density of the periventricular white matter, more often around the anterior horns of the lateral ventricles ("caps", "Mickey Mouse ears");
• many lacunar infarctions (often clinically "silent") in the white matter and subcortical nodes, less often in the pons and cerebellum;
• decrease in the volume of perivascular white matter and expansion of the ventricular system.

The clinical symptoms of SAE [5, 7] are as follows:

• the first signs of the disease in the form of memory loss (initially - forgetfulness), occurring between 55 and 75 years of age;
• the development of the disease on the background of hypertension, which is characterized by: 1) sharp fluctuations in blood pressure with frequent hypertensive crises (often with mild "background" hypertension); 2) violation of the circadian rhythm of blood pressure: an increase or insufficient decrease (less often a sharp decrease) at night before waking up and / or in the first hours after waking up; 3)

hereditary predisposition (hypertension with severe crises, strokes, cognitive impairment in relatives);

• stepwise or gradual progression of cognitive impairments (disorders of attention, memory, visual-spatial perception, poor sensations, less often speech disorders), reaching at the final stage (on average within 5-10 years) the degree of dementia; despite the generally progressive process of increasing cognitive impairment, periods of stabilization ("plateau") and even improvement are possible;

• progressive increase in walking disorders ("walking frontal dyspraxia"): destabilization of the pace and rhythm of movements, disautomation of walking, increased tendency to fall, at the final stage - the impossibility of independent movement;

• progression of pelvic disorders - from periodic urinary incontinence to a complete lack of control over urination, and then over defecation;

• Against the background of progressive cognitive impairment, the majority of patients with SAE develop focal neurological symptoms: 1) paresis of the extremities (usually mild and moderate, in most cases completely regressing), pyramidal signs; 2) extrapyramidal disorders (often parkinson-like akinetic-rigid or amyostatic syndrome); 3) pseudobulbar syndrome (dysarthria, dysphagia, violent crying and laughter);

• as the disease progresses, the progression of emotional-volitional disorders: at first, asthenic, astheno-depressive and neurosis-like syndromes are observed, later - apathy, abulia, loss of interest in the environment, narrowing of the circle of interests, emotional impoverishment;

• in the early stages of the disease, frequent complaints of headaches (migraine-like, tension headaches), dizziness (non-systemic), sleep disturbance.

Multi-infarct hypertensive encephalopathy (MIGE)

MIGE differs from SAE in that the morphological picture of the disease is dominated by a multi-infarction state - the development of many small deep lacunar infarcts in the white matter of the cerebral hemispheres, subcortical ganglia, thalamus, base of the brain bridge, cerebellum, less often in other areas of the brain. MIGE is characterized by:

• acute or stepwise development of neurological symptoms and cognitive impairment;

• detection by computed tomography (CT) or magnetic resonance imaging (MRI) of many small post-stroke cysts (a consequence of repeated lacunar infarcts, often clinically "silent"), combined with moderate brain atrophy and expansion of all parts of the ventricular system, in the absence or slight severity leukoaraiosis.

Risk factors for the development of MGE, in addition to hypertension, are [10]:
• diabetes;
• hyperlipidemia;
• smoking;
• alcohol abuse;
• changes in the rheological properties of blood: hyperviscosity, increased aggregation of platelets and erythrocytes [11].

The clinical picture of MGE is represented by cognitive impairments, which, unlike those in SAE, relatively rarely reach the degree of dementia, pseudobulbar, subcortical, cerebellar syndromes, and the so-called lacunar syndromes. The development and severity of cognitive impairments in MGE are largely associated with the location of infarcts in functionally significant areas: the anteromedial parts of the thalamus, the region of the frontothalamic pathways, and the head of the caudate nucleus [12–14].

Algorithm for diagnosing dyscirculatory encephalopathy

In clinical practice, both overdiagnosis and underdiagnosis of DE are often observed. To make a correct diagnosis, it is necessary, along with obtaining detailed medical history data, a thorough examination of the neurological status and the state of internal organs (primarily the state of the cardiovascular system), to conduct a number of paraclinical research methods, among which the leading ones are neuroimaging, neuropsychological methods and methods of studying blood vessels supplying the brain. The following additional methods of examination are most appropriate:
• neuropsychological research with special attention to the state of memory, attention and intelligence;
• CT or MRI of the brain;
• Ultrasound of vessels supplying the brain: common and internal carotid arteries, subclavian and vertebral arteries and main intracerebral arteries (anterior, middle and posterior cerebral, basilar artery), and if necessary - radiopaque cerebral angiography or magnetic resonance angiography;
• ECG and measurement of blood pressure in dynamics, if necessary, echocardiography, Holter monitoring, daily monitoring of blood pressure;
• study of rheological properties of blood and hemostasis system;
• study of lipid (lipidogram) and carbohydrate (blood glucose) metabolism.

Algorithm for the treatment of dyscirculatory encephalopathy

In the treatment of chronic vascular diseases of the brain, there are three main measures: 1) prevention of progression (or slowing down the progression) of DE, including the prevention of the development of strokes (including repeated ones), which often occur against the background of DE; 2) treatment of the main DE syndromes, improvement of the state of blood circulation and the functional state of the brain, including antioxidant, neurotrophic and vasoactive therapy; 3) rehabilitation, sanatorium treatment.

Preventive measures aimed at preventing or slowing down the progression of DE and the development of stroke are as follows: 1) non-specific prophylactic treatment taking into account risk factors; 2) specific individualized prevention, taking into account the etiology, pathogenesis and form of DE.

As a result of epidemiological studies, a number of unfavorable factors contributing to the development of stroke and DE have been identified. When these factors are present, a person has an increased risk of having a stroke or DE compared to other people of the same age and sex who do not have risk factors. The main curable risk factors for the development and progression of the main forms of DE and the development of stroke (including recurrent) are:

• AH with sharp fluctuations in blood pressure and disruption of the normal circadian rhythm;
• atherosclerosis of the main arteries of the head; carotid and vertebral arteries;
• heart disease with high embologenic potential - atrial fibrillation in coronary artery disease and rheumatic heart disease, endocarditis, etc.;
• diabetes;
• smoking;
• obesity and sedentary lifestyle;
• chronic stressful situations;
• platelet hyperaggregability, increased blood viscosity and hematocrit.

The role of individual factors in the development of different forms of DE is not the same. The main risk factor for the onset and progression of SAE and MIGE is hypertension. General principles for the treatment of hypertension [15]:

• starting treatment with minimal doses of one drug;
• in case of insufficient effectiveness when using large doses of the initially chosen drug or if side effects appear, switching to a drug of another group;
• the use of long-acting drugs (24-hour effect with a single application);
• self-monitoring of patients for the effectiveness of treatment, especially when choosing a dose of the drug (2-fold measurement of blood pressure at home);

• periodic daily monitoring of blood pressure (in hospital and outpatient);
• reduction of salt intake up to 2 g/day;
• taking antihypertensive drugs should be constant;
• decrease in the severity of the rise in blood pressure, often observed in patients with hypertensive encephalopathy, without excessive reduction in blood pressure, which is noted in some patients with hypertensive encephalopathy (which can lead to increased ischemia of the periventricular white matter and deepening cognitive deficits);
• in advanced stages of hypertensive encephalopathy, maintaining systolic blood pressure at 135–150 mm Hg. Art. due to the threat of a decrease in perfusion pressure in the ischemic periventricular white matter.

In addition to adequate antihypertensive therapy, prophylactic treatment for SAE and MGE should include: • antiplatelet therapy [11, 16]; • pentoxifylline (to improve the state of the microvasculature); • vasoactive and antioxidant drugs. Antioxidant therapy Due to the important role played by oxidative stress (activation of lipid peroxidation with accumulation of free radicals in ischemic areas) in the death of the medulla in cerebrovascular diseases, patients with DE are shown courses of antioxidant therapy [17, 18 The vasoactive effect of the drug is to increase cerebral blood flow without the effect of "robbing" ischemic zones, reducing the resistance of cerebral vessels. Cavinton improves brain metabolism by increasing the consumption of glucose and oxygen by the brain tissue, increases the concentration of ATP, enhances the exchange of norepinephrine and serotonin. Against the background of taking Cavinton, cognitive functions improve, dizziness, vestibular disorders and the severity of headaches decrease. The hemocorrective effect of Cavinton is to reduce platelet aggregation and blood viscosity, increase the deformation of erythrocytes, which positively affects the state of the microvasculature. Cavinton and Cavinton forte in DE are used according to the following scheme: infusions of Cavinton's solution are prescribed intravenously, up to 80 drops per minute, starting therapy with 20-25 mg, then 30; 40; 50; 50; 50 and 50 mg for 7 days with the transition to Cavinton forte 1 tablet (10 mg) 3 times a day for 11 weeks. It is recommended to repeat the course of therapy after 6 months. Treatment of Patients with Cognitive Impairments The main core of the clinical symptoms of SAE and MIGE are cognitive impairments. Traditionally, piracetam, pyritinol, peptides are prescribed to improve cognitive status. For the past two decades, drugs originally proposed for the treatment of diseases have been successfully used to treat such patients.

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Rehabilitation of patients with dyscirculatory encephalopathy

Rehabilitation is a set of measures aimed at restoring (full or partial) impaired functions, mental or social readaptation of the patient. The general principles of rehabilitation of patients with DE include [5]: • proper organization of work and rest, exclusion of night shifts and long business trips; • moderate physical activity, remedial gymnastics, dosed walking, visiting the swimming pool; • Diet therapy (limiting the intake of salt, animal fats, limiting the total caloric content of food, introducing fresh vegetables and fruits, wholemeal flour products, fish products into a constant diet); • restriction of alcohol consumption (perhaps a daily intake of 200 ml of dry red wine); • climatotherapy at local resorts, in the conditions of low mountains (for example, at the resort of Mineralnye Vody), at sea resorts (in the non-hot season); • balneotherapy, which has a positive effect on central hemodynamics, contractile function of the heart, the state of the immune and autonomic nervous system, which increases emotional tone; the means of choice are radon, brine, carbonic, sulfide, iodine-bromine baths. With pseudobulbar syndrome (dysphagia, dysphonia, dysarthria): • neuromuscular electrical stimulation of the muscles of the pharynx, larynx and tongue using special electrodes (duration of the procedure - 7-10 minutes, periods of stimulation lasting 20-30 seconds alternate with pauses for 1 minute) [24]; • with dysarthria - gymnastics and massage of the muscles of the pharynx, pharynx and articulatory muscles, classes with a speech therapist; • with violent laughter and crying - amantadine (midantan) or PK-Merz 0.2 g per day (in 2 divided doses) for several months. With frontal dyspraxia of walking and walking disorders associated with vestibulocerebellar syndrome, often combined with imbalance: • special therapeutic gymnastic exercises, training in walking on sand and gravel; • to improve the balance function, the use of the biofeedback method according to the stabilogram with the help of computer-stabilographic complexes, which include: a rigid dynamic platform, a computer, a package of applied programs containing rehabilitation games. With motor disorders (with DE, these are usually mild and moderate mono- and hemiparesis), therapeutic exercises are prescribed. Due to the progressive nature of DE, rehabilitation courses must be repeated periodically (the frequency and duration of repeated courses are determined by the patient's condition) in a rehabilitation hospital (in its absence, in a neurological hospital), a sanatorium (preferably a rehabilitation one) and / or outpatient.

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