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DYSCIRCULATORY ENCEPHALOPATHY (REVIEW)

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Abstract
Under the heading "Cerebrovascular diseases" there are diseases similar in clinical and pathogenetic characteristics to DE: I67.2 - cerebral atherosclerosis, I67.3 - progressive vascular leukoencephalopathy (Binswanger's disease), I67.4 - hypertensive encephalopathy, I67.8 - cerebral ischemia (chronic). Unfortunately, the real prevalence and incidence of DE in our country is still unknown. The term cerebrovascular disease, which came to us from abroad (from the English cerebrovascular disease) is not entirely successful, because. in the domestic neurological school, it is customary to name diseases in a pathogenetic sequence. Undoubtedly, the term cerebrovascular disease is more logical, emphasizing the primary role of vascular pathology and hemodynamic disturbances, which subsequently leads to acute or chronic cerebral ischemia.

Keywords: dyscirculatory encephalopathy, cerebrovascular diseases, atherosclerotic, chronic cerebrovascular ischemia, doppler ultrasound.

From the point of view of modern neurological concepts, stroke and dyscirculatory encephalopathy can be characterized as two main clinical syndromes of acute and chronic vascular lesions of the brain, which are the outcome of various pathological conditions of the circulatory system. Disclosure of pathogenetic heterogeneity (heterogeneity of immediate causes and pathogenetic mechanisms of development of acute cerebral ischemia) of ischemic stroke has become one of the main achievements of recent decades in angioneurology. In the 90s of the last century, under the leadership of Academician N.V. Vereshchagin, the doctrine of pathogenetic subtypes of ischemic stroke was created, which differ in etiopathogenesis, clinical picture, nature and rate of recovery, and treatment tactics [4]. Undoubtedly, with the accumulation of our knowledge about the regularities of the functioning of cerebral circulation in normal and pathological conditions, the role of cardiac, hemostatic, endothelial and immunological factors in maintaining adequate cerebral hemodynamics, the number of such pathogenetic subtypes will increase. Institute (now the Scientific Center) of Neurology of the Russian Academy of Medical Sciences, under the leadership of Z.A. Suslina, continuing the teachings of N.V. Vereshchagin about the heterogeneity of cerebral ischemia, identifies the following main

pathogenetic subtypes of ischemic stroke: atherothrombotic - 34%, including thrombosis of cerebral vessels (21%), arterio-arterial embolism (13%); cardioembolic - 22%; hemodynamic - 15%; lacunar - 22%; stroke according to the type of hemorheological microocclusion - 7% (% reflects the frequency of occurrence of various strokes in the neurological departments of the Institute of Neurology of the Russian Academy of Medical Sciences) [5].

Chronic forms of cerebrovascular diseases and, in particular, dyscirculatory encephalopathy, despite their significant prevalence in outpatient practice, are in the shadow of studying various types of strokes in modern publications. The modern paradigm of heterogeneity of cerebral ischemia in stroke is fully applicable to the assessment of the polymorphic clinical picture of DE, the definition of differentiated treatment tactics, taking into account the pathogenetic variants of chronic cerebral ischemia. Starting from the classical scientific works of E.V. Schmidt and N.V. Vereshchagin, to date, most publications distinguish 4 main pathogenetic types of DE: atherosclerotic, hypertensive, venous, mixed (with a combination of several etiological factors) [2, 3, 6].

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Some authors distinguish the following main variants of DE:

1) hypertensive (subcortical arteriosclerotic encephalopathy and hypertensive multi-infarct encephalopathy);
2) atherosclerotic;
3) chronic vascular vertebrobasilar insufficiency;
4) mixed forms [1, 7].

Separate publications are devoted to various additional factors, often of independent pathogenetic significance, leading to the development of chronic cerebral ischemia: angiospasm, repeated microthrombosis, microembolism, microhemorrhage, antiphospholipid syndrome, diabetes mellitus, hyperhomocysteinemia, vasculitis, transient bradyarrhythmia, hemostatic activation, hemorheological disorders,

endothelial dysfunction [6, 8, 9]. Until now, there is no generally accepted classification of pathogenetic variants of DE. The main domestic neurological schools came to a consensus only on two problems - the etiology of DE and the characteristics of the severity of the pathological process. Depending on the etiology, the following types of DE are distinguished: hypertensive, atherosclerotic, venous, and mixed. The staging of the development of the pathological process is characterized by the allocation of three stages of DE (I - moderately pronounced, II - pronounced, III - pronounced), depending on the severity of clinical manifestations and structural changes in the brain substance [1, 6].

Arterial hypertension is now recognized as a pandemic that determines the structure of morbidity in most countries of the world, including the Russian Federation. According to a survey of a representative sample, the age-standardized prevalence of arterial hypertension in Russia is 39.2% among men and 41.1% among women [10]. In Russia, cardiovascular diseases account for 56.5% of all deaths, while the absolute mortality rates from cardiovascular diseases are currently 5-6 times higher in our country than in Western countries [11]. Among all risk factors leading to chronic cerebral ischemia and vascular dementia, arterial hypertension occupies a leading position in our country [12].

The main nosological form of hypertensive encephalopathy is subcortical arteriosclerotic encephalopathy (SAE), which was described in 1894 by the German neurologist Otto Binswanger. The disease is characterized by progressive vascular damage to the white matter of the brain, leading to dementia. According to the literature, the main etiological factors in the development of SAE include the following: arterial hypertension (in 95-98% of cases), amyloid angiopathy, cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL) - cerebral autosomal dominant angiopathy with subcortical infarcts and leukoencephalopathy [1, 13].

In scientific papers on hypertensive encephalopathy, the following main risk factors for the development and progression of SAE leading to the development of dementia are indicated: by 10-22%), increased nighttime blood pressure, a sharp decrease in nighttime blood pressure by more than 22%; 2) increased hematocrit (more than 45%); increased fibrinogen levels, platelet and erythrocyte aggregation, blood viscosity; 3) a characteristic neuroimaging picture in the form of widespread periventricular leukoaraiosis (translated from the Greek "leuko" - leukos - "white", "areosis" - araiosis - "discharge") in combination with lacunar infarcts [1, 13, 14, 15]. Most authors recognize that the basis of the pathology of the white matter (myelinated pathways) of the brain in SAE is arteriosclerosis of arterioles and small arteries with

a diameter of less than 150 μm, which is expressed in hypertrophy of the media, hyalinosis, a sharp thickening of the vascular wall with a narrowing of the lumen of the artery [7] . Some researchers note that the severity of leukoaraiosis is an independent predictor of the development of dementia in patients with cerebrovascular diseases [16, 17]. According to N.N. Yakhno et al., the severity of leukoaraiosis determines a decrease in cognitive functions, especially of a neurodynamic and regulatory nature, when its area exceeds 10 cm2 at the level of the lateral ventricles, with a predominant lesion of the frontal lobes [18]. The basis of the clinical manifestations of SAE are: 1) memory loss followed by a gradual or stepwise increase in cognitive disorders, turning into vascular "subcortical" dementia; 2) a progressive increase in walking disorders, mainly of the "frontal" type with frequent falls; 3) focal neurological syndromes (pyramidal, extrapyramidal, pseudobulbar); 4) dysfunction of the pelvic organs with impaired control of urination, and then defecation; 5) emotional-volitional disorders and sleep disorders [7, 13].

The second nosological form of hypertensive DE is multi-infarct hypertensive encephalopathy (MGE), which is characterized by: acute or step-like development of neurological symptoms and cognitive deficit, neuroimaging (identified by MRI/CT) signs of multiple post-stroke lacunar cysts (1-15 mm in diameter) in combination with moderate hydrocephalus replacement and mild leukoaraiosis [1, 14]. According to the literature, in most cases, hypertensive lacunar infarcts are asymptomatic, in particular, MRI scans reveal 10 times more “silent” lacunar infarcts than those with clinical manifestations of a stroke. An increase in the number of "silent" lacunar cerebral infarcts is a predictor of the development of cognitive impairment, in particular, the risk of developing vascular dementia becomes 2 times higher [19]. The clinical picture of MGE is based on the following main syndromes: cognitive impairments that rarely reach the level of dementia, speech disorders, focal neurological syndromes (motor monoparesis, pseudobulbar, extrapyramidal, pyramidal, cerebellar), walking disorders [14].

Atherosclerotic DE is a complex of diffuse and focal changes in the brain of an ischemic nature, caused by atherosclerosis of the cerebral and extracranial arteries. Interrelated processes in atherosclerotic angioencephalopathy include: the formation of atherosclerotic plaques with destructive and reparative changes in them, stenosis characteristic of atherosclerosis (atherostenosis), closure of the lumen of the artery with a plaque (atheroobliteration), embolism with the material of a disintegrated plaque (atheroembolism), thrombosis in the area of the plaque and behind it outside, secondary structural changes in blood vessels associated with hemodynamic disorders [2, 4].

Clinical manifestations of atherosclerotic DE depend on the following factors: the degree and rate of formation of arterial stenosis, the structure of the atherosclerotic plaque (hypoechoic, unstable, complicated), atherosclerotic lesions of a certain basin (carotid, vertebrobasilar), the presence of echeloned extra-intracranial "tandem" stenoses, the condition collateral circulation, resistance of the brain parenchyma to hypoxia, the state of general hemodynamics. The morphological basis of atherosclerotic DE is: granular atrophy of the cerebral cortex with small superficial infarcts, multiple atherosclerotic lacunar subcortical or cortical-subcortical infarctions, and enlargement of the subarachnoid spaces and cerebral ventricles [18]. According to the literature, the basis of the clinical manifestations of atherosclerotic DE are the following syndromes: 1) progressive cognitive decline, rarely reaching the degree of dementia ("cortical" vascular dementia); 2) disorders of higher cortical functions (speech, reading, writing, gnosis, praxis); 3) focal neurological syndromes depending on the location of cerebral infarctions (monoparesis, monoanesthesia, ataxia, vestibulopathy, dysfunction of cranial nerves); 4) emotional-volitional disorders [1, 20].

At diagnosis, in the process of outpatient management of patients with clinical manifestations of chronic cerebrovascular ischemia, vertebrobasilar insufficiency (VBI), often referred to as "DE, predominantly in the vertebrobasilar basin (VBB)", is widespread. Often the reason for making this diagnosis is the patient's complaints of dizziness, tinnitus, unsteadiness and impaired coordination, as well as changes in hemodynamics in the VBB detected using Doppler ultrasound.

Insufficient awareness of outpatient physicians in determining the nosological affiliation of the vestibular syndrome leads to overdiagnosis of VBI, which ultimately results in the unreasonable prescription of vasoactive and nootropic drugs. The term "dizziness", which refers to the sensations of an imaginary movement of objects or one's own body in space, or a distortion of gravitational orientation, often has an unreasonably extended use [15]. In the US, dizziness accounts for 3.3-4% of the reasons for seeking emergency medical care. Among the main causes of vertigo that forced 9472 patients to seek emergency care, 32.9% of cases were diseases of the inner ear, 21.1% - cardiovascular, 11.2% - neurological (including 4% cerebrovascular), 11 % - metabolic, 7.2% - mental disorders [19]. Among the most common causes of vestibular syndrome not associated with VBN, the following diseases are described in the literature: labyrinthitis, neuritis of the cochleovestibular (VIII) cranial nerve, benign positional paroxysmal vertigo, Meniere's disease, migraine vertigo (vestibular migraine), orthostatic arterial hypotension, asthenic syndrome, neurotic disorders

(depressive, anxiety, somatoform), metabolic disorders (anemia, hyperglycemia, electrolyte imbalance) [21, 23].

In modern social conditions, which are characterized by an increase in psycho-emotional and informational loads, neurotic and psychosomatic diseases are becoming more common. Against the background of low availability of psychotherapeutic assistance, functional disorders of the central nervous system lead to autonomic dysfunctions, in particular, to cerebral autonomic paroxysms (diencephalic crises) that mimic VBI or vestibulopathy. Diencephalic crises are often the reason for overdiagnosis of cerebrovascular disease, which leads to erroneous tactics of using vasoactive and nootropic drugs that worsen the condition of patients with neurotic and vegetative disorders.

Ultrasound methods for studying cerebral circulation (Dopplerography, duplex scanning) are becoming more available, and are increasingly used in examining a patient with clinical manifestations of cerebrovascular insufficiency. The conclusions of ultrasound diagnostic specialists are replete with signs of extravasal spondylogenic effects (compression) on the vertebral arteries in the V2 and V3 segments, which is often the reason for the diagnosis of spondylogenic vertebral artery syndrome (spondylogenic VBI, posterior cervical sympathetic Barre-Lieu syndrome). A large number of scientific papers on spondylogenic cerebral disorders and the frequently encountered ultrasonic phenomenon of extravasal effects on vertebral arteries ultimately lead to unjustified overdiagnosis of cervicogenic cerebral syndromes, low efficiency in the treatment of vestibular syndrome, and a decrease in the differential diagnostic activity of outpatient doctors. Unfortunately, the erroneous tactics of managing patients with VBN syndrome, vestibulopathy, migraine are still widespread, the treatment of which is dominated by methods of vertebroneurological rehabilitation (massage and manual therapy on the cervical-collar region, osteopathy, wearing a Shants collar), instead of individually selected therapy of the main non-spondylogenic disease. Due to the lack of data on the specificity of symptoms and signs of the Barre-Lieu syndrome based on the principles of evidence-based medicine, in modern English-language literature there is often an opinion that its existence, as well as the cervical vertigo syndrome, is not proven [21, 24, 25].

The lack of clear ideas about a wide range of pathogenetic variants of chronic cerebral ischemia, the vagueness of the criteria for assessing the stage of dyscirculatory encephalopathy, lead to frequent diagnostic and therapeutic errors in the outpatient practice of neurologists and family doctors. The most optimal in clinical work are the criteria for diagnosing DE, described by M.M. Identical in 2007 [6]:

The presence of complaints and clinical picture characteristic of a cerebrovascular disease.

The presence of signs of damage to the cerebrovascular bed (according to Doppler or duplex scanning):

a) stenosing (occlusive) processes;

b) functional circulatory disorders (blood flow asymmetry, changes in the range of cerebrovascular reactivity).

3. Presence of signs of morphological changes in the medulla (according to neuroimaging data: MRI, CT):

a) diffuse atrophic changes in the form of expansion of the ventricular system and / or subarachnoid spaces;

b) focal changes in gray and white matter in the form of postischemic cysts, lacunar strokes;

c) subcortical and periventricular leukoaraiosis.

4. Presence of signs of cardiovascular disease and / or angiopathy.

5. Availability of laboratory data on changes in blood composition:

a) violation of lipid metabolism;

b) violation of the rheological and coagulant properties of blood;

c) changes in specific indicators (homocysteine, LE-cells, S-protein, etc.).

According to the author, the diagnosis of DE is established in the presence of three or more of the five signs and confirmation of causal relationships. In the diagnosis, it is necessary to indicate the etiology of DE and its stage [6].

Diseases is one of the most controversial sections of modern angioneurology. It is advisable to single out the following main areas of therapeutic and preventive measures used in the therapy of DE: 1) prevention of the development and progression of chronic cerebral ischemia; 2) treatment of the main DE syndromes, taking into account their etiopathogenesis and individual characteristics; 3) prevention and treatment of vascular dementia; 4) complex rehabilitation taking into account polymorbid cardiovascular pathology [7].

Prevention of DE is an integral part of general measures to prevent and slow down the progression of cardiovascular diseases. The modern strategy of primary and secondary prevention of stroke is fully applicable to the prevention and treatment of chronic cerebrovascular insufficiency, as a result of which there is no clear line between these two main areas of outpatient angioneurological care. Early detection and timely correction of risk factors for cardiovascular disease and stroke is a key element in the prevention of DE [7]. Classical ideas about the significance of the main risk factors for cerebrovascular diseases, proposed by the American National Stroke

Association in 1991, remain relevant at the present time [6]. The main directions for the prevention of acute and chronic cerebral ischemia are the normalization of the following correctable risk factors: arterial hypertension, hypercholesterolemia and atherosclerosis, heart disease with a high embologenic potential, increased platelet aggregation and blood viscosity. This section of care for patients with DE can be successfully implemented by both a neurologist and a family doctor. In most modern domestic and foreign works on the prevention of cerebrovascular diseases, three main groups of drugs are recommended: antihypertensive agents (angiotensin-converting enzyme inhibitors, diuretics, calcium channel blockers), statins (simvastatin, atorvastatin, rosuvastatin), antiplatelet agents (acetylsalicylic acid , clopidogrel, dipyridamole) [3, 9]. In the case of verified cardioembolism, embolic lacunar infarcts of the brain, risk of cardioembolic stroke, many authors recommend indirect anticoagulants (warfarin) [13, 20].

The high efficiency of angiosurgical treatment of extracranial artery pathology in the prevention of ischemic stroke has been successfully demonstrated in large multicenter foreign studies (NASCET, ESCT, ACAS, SPACE, EVA-3S, SAPHIRE, CAVATAS, etc.) [20, 31]. Since most scientific studies have evaluated the effectiveness of carotid endarterectomy and stenting in the prevention of ischemic stroke, it is absolutely justified in modern conditions to expand the range of positive effects of operations on the carotid and vertebral arteries, in particular, the prevention and treatment of atherosclerotic DE. The reperfusion effect of endarterectomy and stenting is especially relevant in patients with combined stenosis of the carotid and vertebral arteries. This type of surgery on extracranial arteries is an important pathogenetic method for preventing the development of vascular dementia.

According to academician A.V. Pokrovsky, about 130,000 carotid endarterectomies are performed annually in the United States, and only about 3,000 in Russia [43]. According to the Scientific Center for Cardiovascular Surgery. A.N. Bakulev in Russia, about 2500 surgical interventions on extracranial arteries are performed annually, which is 17 surgical interventions per 1 million population. If we use the estimated standard for the minimum need for such operations, developed in the United States (750 per 1 million population), in the Russian Federation it is necessary to perform 110-120 thousand operations per year [24]. In St. Petersburg and the Leningrad region, about 400-450 operations on extracranial arteries are performed annually. Given that St. Petersburg remains one of the “oldest” cities in Russia in terms of age composition, while the prevalence of atherosclerotic lesions of peripheral arteries is significantly progressing, the real need for surgical operations on the carotid and

vertebral arteries is 3500-4500 operations annually, that is, it is covered by about 10%.

Based on the analysis of modern literature, among the recommendations for determining indications for open surgery (endarterectomy) on extracranial arteries, the following can be distinguished: 1) stenosis of the internal carotid artery (neurologically symptomatic, i.e. acute cerebrovascular accident in history) more than 70%; neurologically symptomatic stenosis of less than 70% in the presence of an ulcerated (embologenic) atherosclerotic plaque; neurologically asymptomatic stenosis over 80%; 2) neurologically symptomatic stenosis of more than 70% or occlusion of the vertebral artery; 3) neurologically symptomatic pathological tortuosity of the carotid and vertebral arteries [20, 25].

The main indications described in major scientific publications for transluminal carotid balloon angioplasty with stenting are as follows: 1) hemodynamically significant intracranial stenosis of the cerebral arteries; 2) multifocal atherosclerotic lesion with the need for simultaneous operations in several places; 3) when the plaque is located in hard-to-reach places (ICA siphon); 4) the presence of severe concomitant pathology; 5) if repeated surgical treatment is required after open surgery [1, 6].

Established for decades and become classic, the ideas of domestic neurologists regarding the treatment of chronic cerebrovascular diseases imply two main areas of therapy: cerebroprotection and reperfusion [5, 18]. Most of the vasoactive and cerebroprotective drugs used in our country are not known abroad. The effectiveness, safety and expediency of their use does not meet the requirements of evidence-based medicine, and is not confirmed by the results of large randomized controlled trials.

The largest number of publications confirming the effectiveness and expediency of using drugs in DE in the domestic literature is devoted to the following groups of cerebroprotective drugs: 1) pyrrolidone nootropics (piracetam, nootropil, lucetam); 2) neurotrophic drugs (cerebrolysin, semax, cortexin, actovegin); 3) drugs that improve cholinergic neurotransmission (gliatilin, citicoline, cereton); 4) substances that affect the GABA system (phenotropil, pantogam, picamilon, phenibut); 5) antioxidants (mexidol, vitamin E, thioctic acid, cytoflavin); 6) preparations of Ginkgo Biloba (EGB 761, tanakan, memoplant) . To date, the following cerebroprotective agents have the highest level of evidence: cerebrolysin (level of evidence B), citicoline, gliatilin (level of evidence C)/

The following vasoactive drugs are well known and widely presented in domestic publications and are recommended by many authors for use in both acute and chronic cerebral ischemia: cavinton, pentoxifylline, Wessel due f, cinnarizine, instenon, nicergoline, nimodipine, vasobral [5 , 57,]. From the group of vasoactive drugs, the

most effective in DE, from the point of view of evidence-based medicine, are drugs such as pentoxifylline and cavinton (level of evidence C).

Improving the efficiency of outpatient care for patients with chronic cerebrovascular diseases is possible through the implementation of two main areas that combine domestic and foreign approaches at the present stage of development of angioneurology: 1) the principle of etiopathogenetic therapy and prevention, taking into account heterogeneity and individual diversity in the development of cerebral ischemia; 2) the use of international principles of evidence-based medicine in the formation of treatment tactics and prescription of drugs.

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