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Clinical Features of Dysirculatory Encephalopathy

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Abstract: *the development of DE is manifested not only by an increase in the severity of a particular syndrome, but also by the complication of the development of several syndromes, primarily motor and cognitive. Age has a certain influence on the severity of clinical diseases in patients with DE. Elderly patients are characterized by a great severity and variety of neurological symptoms. Violation of the balance of walking and rest is most common in patients with pseudobulbar syndrome and severe cognitive impairment, which indicates the common pathological basis of these syndromes.*

Keywords: *DE, pastoral and cognitive disorders, cerebrovascular diseases.*

Depending on the severity of the symptoms, DE is divided into three phases. Stage I is dominated by headaches and feelings of heaviness in the head, general weakness, increased fatigue, emotional lability, decreased memory and attention, subjective disturbances in the form of dizziness (often non-systemic). These disorders are accompanied by anisoreflexia, non-gross ataxia, often vestibular nature, signs of oral automatism, decreased memory, and mild but sufficiently persistent objective diseases in the form of asthenia. At this stage, as a rule, the formation of pronounced neurological syndromes does not yet occur (except for Asthenic Syndrome), and with adequate therapy, it is possible to reduce the severity or eliminate individual symptoms and the disease as a whole.

The series of complaints from patients with Stage II de is similar to stage I, although memory impairment, working capacity, dizziness, increased levels of instability while walking, and complaints about headaches and other manifestations of the asthenic symptom complex are somewhat less common. At the same time, focal symptomatology in the form of reflexes of oral automatism, central insufficiency of the facial and hypoid nerves, coordinator and oculomotor diseases, pyramidal insufficiency, extrapyramidal diseases becomes more pronounced, cognitive and emotional disorders are exacerbated. At this stage, it is possible to distinguish certain dominant neurological syndromes – postural and cognitive disorders, pyramidal, etc., which can significantly reduce the professional and social adaptation of patients.

In Stage III of DE, The amount of complaints decreases, which is accompanied by a decrease in criticism of the condition of patients, although complaints about decreased memory, instability during walking, noise and heaviness in the head remain. Objective neurological disorders, such as imbalance during rest and walking, extrapyramidal, pyramidal, pseudobulbar, and cognitive disorders, are significantly more pronounced. The appearance of pronounced pseudobulbar diseases can be associated with damage to the bridge or the nucleus of the serotonergic suture in the area of the pathways connecting this nucleus with the Cerebral Hemispheres. In addition to the foundation of the bridge, damage to the inner capsule and legs of the brain, as well as the pathology of the cerebellum and its connections, are of particular importance. Most often, paroxysmal conditions are observed-falls, fainting, epileptic seizures. What distinguishes this stage from the previous one is that patients with stage De III experience several distinct syndromes, with stage De II being dominated

by one. Patients with DE III Stage are essentially dysfunctional, their social and daily adaptation is sharply disrupted.

Thus, the development of DE is manifested not only by the increased severity of a particular syndrome, but also by the complexity of the development of several syndromes, primarily motor and cognitive disorders. Age has a certain effect on the severity of clinical disorders in patients with de. Elderly patients are characterized by the great severity and variety of neurological symptoms. Violation of the balance of walking and rest is most common in patients with pseudobulbar syndrome and severe cognitive disorders, which indicates the totality of the pathological basis of these syndromes. These disorders are often based on fronto-subcortical separation syndrome. In its pure form, dominance in the neurological state of any disorder is extremely rare. In particular, in patients with de, pronounced pyramidal disorders are associated, in most cases, with a past stroke.

Among Movement Disorders, extrapyramidal and atactic disorders deserve special attention.

It should be noted that vascular parkinsonism accounts for 3-12% of all cases of this clinical syndrome, the occurrence of which in patients with de may be due to heart attacks in the area of basal ganglia and diffuse damage to the white matter of the Cerebral Hemispheres, especially frontal localization. Diffuse white matter changes are based on microinfarcts and demyelination. Parkinsonism of vascular Genesis is characterized by stiffness and bradykinesia, especially in the lower extremities, as well as postural disorders. However, recreational tremors typical of Parkinson's disease are very rare in patients. The appearance of vascular Parkinsonism is more associated with a violation of thalamo-cortical connections with multiple lacunar infarcts than with direct damage to the striatum. As a cause of vascular parkinsonism, isolated foci in the area of Black Matter are extremely rare.

The appearance of postural diseases in vascular Parkinsonism is associated with a violation of the connection between the motor and additional motor cortex – and basal ganglia, as well as direct damage to the basal ganglia. Against the background of prescribing Levodopa preparations, a significant improvement in postural diseases is usually not observed. In vascular parkinsonism, gait disorders are similar to gait apraxia with normotensive hydrocephalus.

Previously, levodopa preparations in vascular parkinsonism were considered ineffective, or their effect was insignificant and short-lived. However, contrary to the existing opinion, it is currently shown that it has a good effect on the background of prescribing levodopa drugs in patients with heart attacks in the area of basal ganglia and diffuse damage to the white substance. In addition, the complications of levodopa therapy in patients with Parkinsonism (for example, in the form of medicinal dyskinesia) are characteristic of the neurodegenerative process and are not a sign that excludes the diagnosis of vascular parkinsonism. With DE, atactic diseases are common, which are manifested by standing and gait disorders. Walking is the result of the interaction of three interconnected brain functions-locomotive, balance (balance) and adaptive reactions. The basis of the locomotive is the consistent implementation of a number of stereotypical contractions (synergies) of the trunk and limb muscles, which leads to a step reaction. At the same time, in order to carry out walking, the structures of the central nervous system must have the ability to start and stop walking, change the gait order (including the speed and acceleration of movement), in order to adapt to changes in the surface on which a person walks, turns and changes in the direction of movement. Maintaining balance (Ballance) involves postural reactions (synergies) needed to stay upright and stay upright while walking. Standing itself is an active process, in which usually the vibrations of the center of gravity do not extend beyond the support area (the base due to the level position of the legs). Waiting for Postural reactions (warning) consists of activating postural muscle groups corresponding to the planned movement to prevent possible balance disorders associated with voluntary movement.

The term " ataxia "(translated from Greek – "not intended") is used to refer to irregular, poorly coordinated, or awkward movements that are not associated with paresis, muscle tone disorders, or violent movements. Ataxia can be manifested by maintaining body balance and gait disorders,

including vibration during walking, coordination and speech disorders, which in their manifestation resemble the speech of a drunk person. One of the most common causes of ataxia is damage to the cerebellum – the main structure of the central nervous system, which provides coordination of movements. Usually, it is involved in the Coordination of movements, including the organization of subtle movements of the fingers, and also regulates the tone of the muscles, which ensures the maintenance of the position. Symptoms that appear when the cerebellum is injured are never limited to a single muscle, muscle group or any particular movement, but are rather more common in nature. In addition to ataxia, when the cerebellum is damaged, muscle hypotension occurs, which is most pronounced in the upper extremities, as well as increased muscle fatigue. Patients with cerebellar lesions often note an exacerbation of atactic diseases against the background of fatigue (sleepless night, excessive physical activity, etc.).

In vascular diseases of the brain, cerebellar symptomatology is usually accompanied by signs of damage to the brain stem, which is understandable given the totality of blood supply to these structures. Infarcts in the cerebellum range from 0.5 to 1.5% of all infarcts, with mortality rates ranging from 20 to 50%. Temporary ischemic attacks in the Vertebral-basilar system can be manifested by sudden episodes of miscarriage (see fig. drop-attacks), which is associated with temporary ischemia of the pyramidal pathways in the trunk area or paramedial parts of the reticular formation. In Anamnesis, these patients, as a rule, have indications of similar attacks, and the corresponding symptoms (ataxia, diplopia, hemiparesis, etc.) are detected in a neurological state.

At the same time, a violation of gait and balance in patients with de can be associated not only, but also in some cases, pathology of the executive branch of the statolomotor system (paresis, cerebellar ataxia, extrapyramidal diseases), but also a defect of the central mechanisms of voluntary movement management. In accordance with the idea of the hierarchical principle of building a Statolomotor system, several levels of gait and balance disorders are distinguished. Violations of the "high level" of regulation of gait and balance include violations of planning and programming communications carried out by frontal structures. "Mid – level" executive structures include the primary motor cortex, pyramidal pathways, and dysfunction of the extrapyramidal system. The "low level" of the formation of gait and balance disorders is associated with damage to the peripheral nervous system and musculoskeletal system.

Most patients with DE have a clinically or subclinically specific defect in the functioning of the static-motor system. In the early stages of DE, The imbalance is represented by a compensatory change in walking parameters. As the disease progresses, the breakdown of the functional statolomotor system occurs. Clinically, this is manifested by a violation of proactive dynamic control and the addition of a defect of the locomotor component, the violation of which manifests itself as one of the main features of the central step generator – a violation of the rhythm of movement. Clinical manifestations of movement rhythm disorders are gait onset disorder ("initiation" of the movement program)," hardening" during walking (breakdown of program execution), and pathological asymmetry of the step. Such disorders are the result of disorders or their connections that modulate the supraspinal effects of cortical-subcortical structures. Thus, there is a breakdown of the static-motor system, which is associated with dysfunction of the software control unit. It should be borne in mind that the peculiarity of brain damage in chronic vascular-brain failure is determined by the specific mechanism that is leading in this patient. Since there are different pathogenetic variants of the disease that are accompanied by different morphological changes (focal and/or diffuse), the clinical diversity of statolomotor diseases in De is quite understandable. In some cases, for example, with the predominance of the cerebellum and its connections, cerebellar ataxia prevails in the clinical picture, in others in the event of a violation of the connections of the cortical sections with subcortical structures – maintaining balance and a violation of high gait. In the extended stages of DE, the latter option occurs more often, which is not surprising, since this is the peculiarity of the disease, especially in elderly and elderly patients, the spread of brain damage, the greatest effect on the ischemic process of subcortical white matter, which connects the frontal cortex with other structures involved in providing statolomotor functions.

Examination of patients with dysirculatory encephalopathy with Anamnestic data and the results of neurological studies are of leading importance for the diagnosis of de. Clinical neurological examination, in addition to traditional tests and a set of samples, should include an assessment of the patient's motor functions. To do this, it is important to assess the speed of walking, the length of the step, the distance between the legs while standing and walking, the presence of difficulties when starting to walk and the ability to turn. In addition, the patient should be asked if he has difficulty getting out of the chair or bed. If the patient has drops, their nature is carefully determined.

To assess his walking, the patient is asked to walk in a straight line, then turn. In case of damage to the cerebellum, characteristic disorders are noted in the form of walking with common legs, pronounced squats and deviations from a straight line. The step length is reduced, the steps themselves are irregular and the length is not the same. It is very easy to determine the irregularities of the steps by asking the patient to walk in a straight line. When walking, the legs often rise excessively high and come into contact with the surface with excessive force. Synchronous movements of the hands are disturbed during walking. For milder disorders, existing disorders can be diagnosed by tandem walking, backward walking, and excluding visual control. In severe cases, the patient cannot walk at all (Abkhazia). Symptomatology, as a rule, is most pronounced when the direction of movement suddenly changes or when trying to start walking immediately after a sharp rise from the chair. The severity of atactic diseases also increases when the patient tries to reduce the step base while walking. An assessment of standing is also necessary.

Neuropsychological examination of patients with DE allows the objectification of cognitive disorders. In a physical examination, great attention is paid to assessing the state of the cardiovascular system. It is necessary to auscultate the main arteries of the head. In patients with noise over the projection of the carotid arteries, carotid stenosis is more than twice as pronounced as in people without such noise. At the same time, about a third of patients with Hemodynamically significant stenosis do not have noise phenomena and a large proportion of noisy people do not have carotid stenosis. Important information can be obtained through daily monitoring of blood pressure and heart rate. Both an increase in blood pressure and a decrease in it are important. The main sign of the diagnosis of orthostatic hypotension is the connection of clinical diseases with the vertical position of the patient. The criterion for orthostatic hypotension is a decrease in systolic blood pressure by 20 mm HG. or diastolic blood pressure-10 mm HG. , when you raise or raise your head more than 60 degrees for three minutes. It should also be borne in mind that after standing, low blood pressure can last much longer – no more than 20 minutes. The severity of orthostatic hypotension can be influenced by a number of factors-the speed of getting up, the time of day (orthostatic hypotension is significantly manifested in the morning), high ambient temperature, alcohol consumption, exercise, etc. The cause of orthostatic hypotension can be yatrogenic effects (the use of anti-parkinsonian drugs, including levodopa, nitrate, etc.) that lead to impaired vegetative sympathetic vasoconstriction. In patients with diabetes mellitus, complicated by the development of autonomic polyneuropathy, prescribing insulin can lead to an increase in transcapillary loss of albumins or a decrease in blood volume due to the vasodilation effect, which increases the existing orthostatic hypotension. Since vascular lesions of the brain can be based on various causes – real vascular diseases (atherosclerosis, amyloid angiopathy, vasculitis, pathological curvature and vascular anomalies), heart diseases (ventricular fibrillation, endocarditis, cardiomyopathy, etc.), pathology of the blood system (hypoxemia, hemoglobinopathy, coagulopathy, etc.), then the tactics of further examination are determined by a specific situation.

Doppler ultrasound can be used to assess the condition of the accessory and intracranial vessels.

DEda neuroimaging methods-computed tomography (CT) and magnetic resonance imaging of the brain (MRI) results are significant. The main neuroimaging manifestations of DE (and vascular dementia) include small and large post-ischemic foci, diffuse white matter alteration (leukoareosis) and brain atrophy, which is manifested by an increase in the ventricular system and an expansion of the Cerebral Hemispheres furrows. The total volume of ischemic foci, their localization and quantity are important. DEda is associated with an increase in the severity of clinical disorders, as well as the

severity of leukoareosis in the anterior parts of the brain and an increase in the ventricular system. However, neuroimaging changes and clinical compatibility are not always observed. MRI looks more sensitive compared to the CT method for detecting heart attacks and diffuse changes in white matter, but using MRI

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