Dyscirculatory encephalopathy: principles of treatment

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ABSTRACT

This article discusses about how a person ages, his or her body ages, the capacity of the cardiovascular system diminishes, hormones are disrupted, the function of all the sensitive analyzers is impaired, digestion of food becomes difficult, age-related changes in the joints and spine make movement difficult. In people over 70 years of age, gross metabolic disorders are observed in 70%, locomotor system disorders in 85%, reduced functionality of the brain (memory, thinking, depression, sleep disturbance) in over 90%. The sharp increase in vascular disease among the younger generation and the consequent development of vascular discirculatory encephalopathy should be taken into account.

Keywords: Dyscirculatory encephalopathy, dysmnesic, haemodynamics, actovegin and antihypoxant, paroxysmal states, subarachnoid expansion, neurological symptoms

Currently, cerebral vascular disease is a major medical and social problem. This pathology has become the cause of temporary disability and the main cause of primary disability. Acute and chronic forms of cerebrovascular disease are conventionally divided, although in many cases it is impossible to draw a clear line between them and stroke is usually only a certain stage of the development of chronic cerebrovascular disease.

The consequences of these diseases have a negative impact on the country's economy and society in general, and reduce the quality of life of patients and their families. At the same time, the economic losses due to stroke are significant. Most costs (up to 80%) are associated with the cost of hospital treatment and subsequent early and late rehabilitation after stroke. All this points to the importance of systematic analysis and planning of a set of therapeutic and preventive measures for this category of patients. An increasing proportion of the population consists of older people, the "elderly". It is unfair and unjustified to treat the problem of aging as a natural process that does not require the intervention of specialists.

A study of the combined use of neuroprotective therapy with Ceraxon and Actovegin in the treatment of dyscirculatory encephalopathy. Twenty patients aged 40-65 years were examined. The complex therapy was more effective compared to monoprotection due to the restoration of neurological deficits, restoration of the functional activity of the brain and improvement of cognitive functions. For the clinical characterization of brain dysfunction developing as a result of vascular disorders, the concept of "discirculatory encephalopathy" (DE) is widely used in our country.

Dyscirculatory encephalopathy is a slowly progressing disorder of blood supply to the brain, developing most often against the background of atherosclerosis, hypertension, sometimes - against the background of diabetes, syphilis and other diseases affecting the blood vessels of the brain. It is important to note that DE is the result of diffuse multifocal brain damage. In most cases, DE can be confirmed by

various neuroimaging modalities. In CT, DE is characterised by subarachnoid expansion ("atrophy"), periventricular luminescence, and ventricular dilatation, while MRI in T2-weighted images usually shows fine-point multiple foci localised in the white matter. The main etiological causes are atherosclerotic, hypertensive, mixed and venous DE. There are three stages of DE based on clinical signs. Stage I is dominated by subjective disorders such as headaches, sensations of heaviness in the head, general weakness, fatigue, emotional lability, impaired memory and attention, dizziness (more often of a non-systemic nature), unsteadiness when walking, and sleep disturbances. Focal neurological symptoms in this stage are manifested by reflexes of oral automatism, weak convergence of the eyeballs, and sometimes anisoreflexia. Stage II of DE differs from the first in the more persistent and pronounced symptoms and the appearance of signs of pyramidal and extrapyramidal insufficiency. The dominant neurological syndrome - discoordination, pyramidal, dysmnesic, etc. - is characteristic. At stage III, against a background of clear focal neurological manifestations, intellectual and mental disorders become clinically significant and sometimes reach the degree of psychoorganic syndrome.

More often paroxysmal states - falls, fainting, epileptic seizures are observed. Movement and cognitive disorders are considered to be the core of the clinical picture of DE and its distinctive feature. Severe movement disorders are usually associated with an acute cerebral circulation disorder. Progressive extrapyramidal syndrome is another cause of severe movement disorders. Sometimes the patient's motor activities are impeded by an increasing lack of coordination of movements. Along with the progression of focal neurological symptoms as DE progresses, the destruction of higher brain functions occurs. Dementia is a frequent outcome of progressive cerebral blood supply disorders. Successful treatment of DE involves a comprehensive approach to the problem, taking into account compensation for cardiovascular abnormalities, restoration of microcirculation and application of the principles of metabolic therapy.

The subjects of my study were 20 patients aged 40-65 years (13 men and 7 women). The criteria for the analysis of the conducted treatment were the degree of recovery of neurological deficit, regression of the general cerebral symptoms, improvement of the mental status. Additional methods of investigation were used: general laboratory tests, ocular fundus examinations, EEG, CT, MRI of the brain.

Successful treatment of dyscirculatory encephalopathy involves a comprehensive approach to the problem, taking into account compensation of cardiovascular abnormalities, restoration of microcirculation and application of the principles of metabolic therapy. Influence on the metabolic processes of ischemic brain tissue is one of the leading directions of therapeutic tactics in DE. The possibility of influencing such important processes as oxidative damage of the cell, membrane permeability, restoration of energy capabilities looks extremely attractive. It is only necessary to remember that the implementation of therapeutic efforts aimed at restoring the metabolism of nervous tissue is possible only with the restoration of central hemodynamics and elimination of microcirculatory disturbances. Metabolic therapy in DE can have different points of application. Widely used in practice are antioxidants: vitamins A, E, C in various combinations or in complex, actovegin, etc. The inclusion of phospholipid complexes in treatment programs should be considered an indispensable condition for the successful treatment of DE. Phospholipids are necessary for the construction of cell membranes imielin. Traditional "activators" of neural tissue metabolism such as piracetam and others demonstrate high efficacy. In recent years, combined drugs, which form an optimal combination of components influencing the metabolism of neurons, have been actively used, such as Fezam. It is a combined preparation containing 400 mg of piracetam and 25 mg of cinnarizine. The combination of the two components increases the antihypoxic effect and decreases the tone of the smooth muscle of

cerebral vessels. The drug has moderate anti-aggregant activity, stimulates metabolic processes in CNS and increases integrative capacity of the brain. Fezam has significantly fewer side effects than its individual components and is ideal in those cases where the use of piracetam alone causes tenseness and insomnia, and cinnarizine - drowsiness. In this regard, it can be recommended for the working group of patients, as driving and operating machinery is allowed during treatment with Fezam.

A promising direction of metabolic therapy is associated with the use of neurospecific peptides. Low molecular weight peptides (cerebrolysine, cytamines) derived from brain tissue have multidirectional effects in chronic cerebral ischaemia. They contribute to the restoration of inter-neuronal connections, exhibit a stimulating effect, contribute to the restoration of lost functions, activation of disturbed inter-neuronal connections. The use of regulatory peptides in DE should be considered quite reasonable. Of the regulatory peptides already used in clinical practice, melatonin should be mentioned.

Melatonin is a low molecular weight peptide, a product of pineal gland secretion. Its biological effect goes far beyond the regulation of the day-night cycle. It influences the circadian rhythms of BP and takes part in the adaptation of the neuroendocrine system to changing environmental conditions. Thus, the analysis of modern possibilities of DE treatment shows that in most cases these possibilities are not used to the full extent. Simplified and standardized prescription of "vasodilators" and nootropics in most cases does not lead to sustainable improvement of patients' condition. At the same time, the abundance of drugs of different groups that are potentially useful in the treatment of DE should not lead the physician to polypragmasy. It is reasonable to divide the entire treatment cycle into three periods:

1) stabilisation of central, cerebral haemodynamics and microcirculation;
2) application of antioxidants, neuroprotective vitamins, phospholipid and peptide complexes;
3) application of regulatory peptides and nootropics.

Effective treatment of DE can be only with long-term use of complex regimens, taking into account the data of additional studies, including indicators of central and cerebral hemodynamics, laboratory tests reflecting the process of metabolism and hemostasis. Cognitive function and motivation must be maintained in order to carry out active rehabilitation measures. Numerous studies have demonstrated the positive effect of actovegin and ceraxon on these processes. In addition, actovegin is known to stimulate the energy processes of functional metabolism and anabolism with increased energy expenditure, which takes place in restorative kinesitherapy sessions.

Given that the post-stroke period is usually accompanied by progression of cerebrovascular disease, administration of actovegin to stroke survivors is indicated for both therapeutic and prophylactic purposes. It has been observed that basic therapy (antihypertensive and antiaggregant) is not sufficient to prevent the progression of vascular encephalopathy. Additional administration of Actovegin and Ceraxon changed the situation

- Asgenic syndrome, vestibular ataxia, motivation, anxiety, pseudobulbar and amiostatic syndromes were significantly reduced. A statistically significant decrease in the signs of venous dyscirculation was detected. It should be noted that it is the venous vascular component that is of great importance in the development of chronic ischaemia. The presence of a therapeutic "trace" effect by the 3rd month of observation is especially emphasized, which allowed to recommend taking Actovegin by courses of 25 days 2 times a year in a dose of 160 mg (4 ml). The analysis of the literature shows that actovegin and ceraxon can be used at different stages of stroke treatment. Thus, the use of actovegin and ceraxon, influencing such important pathological links of stroke as hypoxia, oxidative stress and energy deficit, is pathogenetically justified and clinically proven at different stages of stroke treatment.

Actovegin as an antihypoxant affects the first stage of the pathological cascade initiated by ischemic cerebral process (promotes cell

survival in ischemic conditions), as an antioxidant reduces pathological effects of primary and repeated wave (after reperfusion) oxidative stress, fills the energy deficit, providing recovery processes, prevents development and reduces severity of cognitive disorders in long-term sequelae of stroke. Continuity in the treatment of patients at different stages of cerebral vascular disease is facilitated by the availability of different medication forms and doses of the drug.

Dyscirculatory encephalopathy is a syndrome of focal or diffuse brain damage, manifesting as a range of disorders: clinical neurological, non-neuropsychological, in some cases psychiatric, and is associated with chronic cerebral vascular insufficiency and/or acute cerebral circulation disorders. The occurrence of acute episodes of cerebral dyshemia can exacerbate the course of DE. The term 'dyscirculatory encephalopathy' reflects both the pathogenetic and morphological features of cerebral damage caused by cerebrovascular disease. The pathogenetic mechanisms that can lead to the occurrence of DE are very diverse.

are manifold. These include chronic vascular cerebral insufficiency, acute cerebrovascular events, and so-called "uncompleted strokes". The term 'incomplete stroke' refers to the area surrounding the infarction zone that has different characteristics (morphological and pathogenetic) from the necrosis. It is the zone of incomplete stroke that is often associated with hopes for effective therapy, both acute and chronic cerebrovascular disorders.

The second part of the term DE, 'encephalopathy', reflects a number of changes that used to be identified mainly on autopsy. Nowadays, with the introduction of neuroimaging techniques, these changes can be assessed in vitro. They are postischemic cysts of different localization, diffuse changes in white matter (leukoareosis), as well as cerebral atrophy. Leukoareosis is often visualized in patients with DE by computed tomography, or better yet, by magnetic resonance imaging. Cerebral atrophy is manifested by enlargement of the ventricular system and subarachnoid spaces. It should be noted that cerebral atrophic changes and leukoareosis can also occur in normal aging, as well as in degenerative processes leading to cognitive impairment, such as Alzheimer's disease.

The pathogenesis of the clinical manifestations of encephalopathy is determined by two main factors. The first one is the focal damage of certain cortical and subcortical areas, which is accompanied by the appearance in the clinical picture of a number of rather well-studied syndromes. The second factor is the phenomenon of cortical-subcortical dissociation, which is caused by disruption of the connections of both cortical areas with each other and the cortex with the subcortical structures. There are a number of clinical syndromes that can occur in patients with this phenomenon. Among these, pseudobulbar and postural disorders as well as cognitive dysfunction should be mentioned. Disruption of anterior (frontal) brain connectivity plays an important role in the occurrence of disassociation phenomenon. The main clinical signs of encephalopathy are as diverse as the variants of cerebral lesions of vascular genesis. But still, among them it is possible to distinguish two main types of disorders, often underlying the disability of patients. These are movement disorders and cognitive disorders. Movement disorders are polymorphic in their phenomenology. In particular, there may be pyramidal disorders, not necessarily paresis, as well as extrapyramidal disorders, often in the form of hypokinesia in the lower extremities (trembling is not common in this category of patients). Atactic disorders of complex genesis and pseudobulbar syndrome can also be detected in patients. The undoubted advantage of actovegin and ceraxon is good tolerability, the possibility of long-term use even in relatively high doses. Side effects in the form of allergic reactions (urticaria, oedema, fever), nausea, fever and fatigue are rare.

Conclusion

Thus, the complex application of Actovegin and Ceraxon in the treatment of dyscirculatory encephalopathy produced a positive therapeutic effect in terms of recovery of haemodynamic disturbances, stabilisation of microcirculation, improvement of cognitive

functions and restoration of disturbed functions of the central nervous system.

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