Clinical And Neurological Immunological Features of The Vertebrobasilar System Against the Background of Vertebral Artery Syndrome

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ABSTRACT

For neurologists, it is important to understand that depression can be organic in nature, associated with local or diffuse brain damage. In such cases, emotional disorders are included in the structure of the general neurological deficit associated with brain damage. Circulatory disorders in the vertebrobasilar system (VBS) have attracted the attention of specialists for a long time. They belong to severe and frequent variants of cerebrovascular pathology. According to the Department of Epidemiology and Demography of the WHO, hemodynamic disorders in the vessels of the vertebrobasilar basin account for more than 30% of all cerebrovascular diseases. About 70% of transient cerebrovascular accidents are due to circulatory disorders in the vertebrobasilar system. The article presents a brief review of the literature data on the problem of circulatory failure in the vertebrobasilar system.

Keywords: Vertebrobasilar system, cerebrovascular pathology, hemodynamic disorders

In recent years, more and more attention has been paid to another type of organic depression associated with cerebrovascular diseases. Stroke and chronic forms of cerebrovascular insufficiency are the most common neurological pathology [4]. This causes a significant prevalence of the so-called vascular depression, which probably underlies some subjective neurological disorders characteristic of the initial stages of dyscirculatory encephalopathy [5]. A correct understanding of the nature of the patient's complaints allows us to build a more pathogenetic-based strategy for helping these patients and improve their quality of life.

Widespread, constant growth, high mortality, damage to people of working age, a high percentage of disability among the sick put the problem of vascular diseases of the brain stem localization in a group of socially significant ones. The number of patients with symptoms of chronic cerebral ischemia is steadily growing, amounting to at least 700 per 100,000 population. It is cerebral stroke and progressive cerebral ischemia that are currently the leading causes of disability [6,17,18]. Among survivors after a stroke, only about 1/3 of patients return to work, and 1/3 permanently lose their ability to work, in varying degrees in need of constant care [2]. In addition, most patients with spondylogenic

circulatory disorders in the vertebrobasilar system are at a young age - from 20 to 50 years [3,4]. Emphasizing the importance of the problem, in 2014 the World Health Organization, the International Stroke Society and the World Stroke Federation launched the Global Initiative, in which stroke was declared a worldwide epidemic that threatens the life and health of the population [18]. In the Republic of Belarus, the extremely high medical and social significance of the problem of chronic forms of disorders of the vertebrobasilar circulation is determined by a steady trend towards population aging and an increase in the proportion of elderly people in the population.

Intensive study of various aspects of the pathology of the vertebral arteries began relatively recently. In the 1950s, the syndrome of circulatory insufficiency in IBS was singled out as an independent clinical concept. At the same time, vertebrobasilar insufficiency was defined as "a reversible impairment of brain function caused by a decrease in blood supply to the area fed by the vertebral and basilar arteries" (thus, the ischemic nature and reversible nature of the disorders were emphasized). In 2020

Features of the structure and functions of the vertebrobasilar arterial system and the peculiarity of clinical symptoms during dyscirculation in it led to the allocation in the latest version of the International Classification of Diseases (ICD-X) [10] "syndrome of the vertebrobasilar arterial system" within the framework of "transient transient cerebral ischemic attacks" [attacks] and related syndromes" (ICD-X, G 45.0). If earlier chronic insufficiency of cerebral circulation in IBS was considered in dyscirculatory encephalopathy, then in ICD-X the term "chronic cerebral ischemia" (CCI) was introduced instead of this term. Various forms of pathology of the vascular system of the brain, leading to CCI, are classified in the ICD in the section "Cerebrovascular Diseases" of the ICD as follows: blockage and stenosis of the precerebral (I 65) and cerebral (I 66) arteries that do not lead to cerebral infarction, other cerebrovascular diseases (I 67), cerebral atherosclerosis (I 67.2), hypertensive encephalopathy (I 67.4), cerebral chronic generalized ischemia (I 67.8), consequences of cerebrovascular diseases (I 69).

For the normal course of the metabolism of brain tissue, the constancy of cerebral blood flow is necessary, which ensures sufficient supply of nutrients to the brain: proteins, lipids, carbohydrates (glucose) and oxygen. Stable maintenance of cerebral blood flow at the level of 50-55 ml / 100 g of brain tissue in 1 min. at the level of the hemispheres and 33 ml/100 g of brain tissue in 1 min. at the level of the cerebellum, it is supported by autoregulation of cerebral blood flow, which is carried out reflexively at the level of large vessels due to the adrenergic and cholinergic receptors of their walls with the help of the regulatory mechanism of the carotid sinus and chemical regulation in the vessels of the microvasculature (with an excess supply of O2, i.e. hypocapnia, the tone of precapillary arterioles increases; with insufficient supply of O2 to the brain, hypercapnia, the tone decreases; in conditions of an increase in the amount of carbon dioxide, the sensitivity of microvessels to it increases). It has been established that the degree of the damaging effect of ischemia is determined, first of all, by the depth and duration of the decrease in cerebral blood flow. The area of the brain with the most pronounced oligemia (< 10-15 ml) becomes irreversibly damaged very quickly within 6 minutes. from the moment of development of ischemia (core, or nuclear zone of ischemia). Within a few hours, the central punctate infarction is surrounded by ischemic, but living tissue - the zone of ischemic penumbra, or penumbra, in which energy metabolism is generally preserved, only functional, but not structural changes are noted [7,12,17]. The severity of ischemic changes is also affected by the rheological properties of blood (viscosity, aggregation ability of blood cells, etc.) and the value of perfusion pressure, which is defined as the difference between mean blood pressure and mean intracranial pressure. The critical level of cerebral perfusion pressure is 40 mm Hg; below this level, cerebral circulation decreases and then

stops. The processes that began in the first hours of acute ischemic brain damage, especially when the area of ischemia is extensive, induce and maintain its other "remote" consequences: the reaction of the genome with the inclusion of genetically programmed molecular programs, dysfunction of the astrocytic and microglial cell pools with the development of immune changes and local inflammation in the focus of ischemia, disorders of microcirculation and the blood-brain barrier [7,17].

Slowly progressive diffuse insufficiency of blood supply to the brain tissue causes chronic cerebral ischemia and leads to a progressive deterioration in the functioning of the brain.

In many works, the ischemic process is considered as a universal mechanism that includes the hypoxic cascade of "calcium" cell death. Oxidative stress, shifts in intracellular calcium ion balance, protease activation, and energy deficiency represent a chain of metabolic changes that occur in tissues during ischemia. The key role of the process of programmed neuron death in the regulation of cellular homeostasis in mature brain tissue is assumed [5,7]. There is even more reason to talk about the presence of neuronal apoptosis in cases of non-stroke course of cerebral vascular disease, short-term ischemia with angiospasm or thromboembolism, manifested by reversible or remitting neurological deficit [5,7].

Circulatory hypoxia of the brain, not being identical to the concept of "brain infarction", is a dynamic process and implies the potential reversibility of functional and morphological changes in the brain tissue. This is largely determined by angiogenesis, the natural biological response of tissue to hypoxia and ischemia, modulated by the release of endogenous growth factors. Often this compensatory response to a hypoxic stimulus is insufficient to return perfusion levels to normal. Moreover, chronic hypoxia leads to a decrease in the ability of cells to produce growth factor in response to subsequent episodes of hypoxia and may be partly responsible for inadequate compensatory angiogenesis [3].

At the beginning of the last decade, the phenomenon of "ischemic tolerance" of the brain was described. The essence of this phenomenon lies in the fact that after the onset of a short episode of ischemia (circulatory hypoxia), the resistance of neurons in vulnerable brain formations (hippocampus, neocortex, striatum, etc.) to the subsequent delayed damaging effect of severe forms of ischemia, leading to their death, significantly increases. necrosis or apoptosis. In recent years, the term "ischemic (hypoxic) preconditioning" has been more frequently used in the literature [1,5], which is considered as one of the forms of adaptation of brain cells, heart and other organs to adverse factors (in particular, severe oxygen supply disorders, blood supply, excitotoxicity - hyperstimulation of the glutamatergic system, etc.).

The hypoxic preconditioning effect triggers a cascade of signal transduction mechanisms involving intracellular regulatory systems, the genome, neuromodulatory peptides, and stress proteins that increase the resistance of brain neurons to severe forms of hypoxia. Of great importance in this process is the adaptive activation of the glutamatergic, calcium, and phosphoinositide regulatory systems, early genes, and transcription factors [13].

Thus, hypoxia of the nervous tissue caused by a microcirculation disorder is only a trigger mechanism for the "ischemic cascade" of pathophysiological changes of a metabolic nature, which lead first to anabolic and then to catabolic processes. To systematize the complex hemodynamic and metabolic changes that occur in the brain tissue at various stages of its circulatory insufficiency, a simplified scheme of the "ischemic cascade" is proposed: a decrease in cerebral blood flow; glutamate "excitotoxicity"; intracellular accumulation of calcium ions; activation of intracellular enzymes; increased synthesis of nitric oxide NO and development of oxidative stress; expression of early response genes; "remote" consequences of ischemia (local inflammatory

reaction, microcirculatory disorders, damage to the blood-brain barrier); apoptosis [7].

The alternative choice between the genetic programs of apoptosis and anti-apoptotic protection and the implementation of the mechanisms of necrotic and reparative processes is determined by the level of trophic supply of the brain tissue [12]. In the first minutes of ischemia, the natural protective reaction of the brain is the synthesis of trophic factors and receptors for them. With rapid and active expression of genes encoding neurotrophins (growth factors), cerebral ischemia may not lead to infarct changes for a long time. In the case of the formation of ischemic damage, a high level of trophic factors ensures the regression of the neurological deficit even with the preservation of the morphological defect that caused it.

The vertebrobasilar system (VBS) is formed by two vertebral arteries, extending from the subclavian to the right by 1.4-3.6 cm and to the left by 1.6-5 cm from their beginning and merging at the base of the brain into the basilar artery, located in the basilar groove on the ventral surface of the pons.

Topographically, 4 parts are distinguished in the vertebral artery (see Fig.): pars prevertebralis (prevertebral) (1) - between musculus scalenus anterior and musculus longissimus cervicis before entering the opening of the transverse process of the VI cervical vertebra; pars transversari a (2) - passes through the holes in the transverse processes of the VI-II cervical vertebrae (the so-called canalis arteriae vertebralis); pars atl anti s (3) (leaving the transverse process of the II cervical vertebra, the artery turns laterally and enters the opening of the transverse process of the atlas, goes around its superior articular fossa behind, passes through the posterior atlanto-occipital membrane, dura mater and enters canalis vertebralis); pars i ntracranialis (4) (from the edge of the foramen magnum to the level of confluence with the analo It has now been established that the most common causes of vertebrobasilar circulation disorders are a decrease in blood flow in the system of vertebral arteries due to their occlusion or damage to the nerve apparatus [3,4,8], and in 65% of cases, the violation of the vertebrobasilar circulation is associated with damage to the extracranial parts of the vertebral arteries. hall factors that have a mechanical and reflex effect on the size of their clearance.

References

1. Khodzhieva D.T., Khaidarov N.K. Khaidarova D.K. Correction of asthenoneurotic syndrome with energy corrector cytoflavin // Neurology. - Tashkent, 2013. - No. 3. P.16-19.,
2. Khodzhieva D.T., Khaidarova D.K., Khaidarov N.K., Samadov A.U. Differentiated therapy in the acute period of ischemic stroke // Neurology - 2011. - No. 4. - P. 34.
3. Pitkanen A., Roivainen R., Lukasiuk K. Development of epilepsy after ischaemic stroke. *Lancet Neurol* 2016; 15: 185-197. DOI: 10.1016/s1474-4422(15)00248-3. PMID: 26597090.
4. Djumayevich N. F. Macro-Microscopic Anatomy and Reactive Changes in the Testimonal Appendices under Exposure to Magnesium Chlorate in Postnatal Ontogenesis //International Journal of Innovative Analyses and Emerging Technology. – 2021. – Т. 1. – №. 6. – С. 152-155.
5. Jumaevich N. F. Comparative characteristics of testicular appendages in normal conditions and when exposed to a biostimulator against the background of radiation sickness //International journal for innovative engineering and management research, 2021й. – С. 28-30.
6. Chang C.S., Liao C.H., Lin C.C. et al. Patients with epilepsy are at an in-creased risk of subsequent stroke: a population based cohort study. *Seizure* 2014; 23: 377-381. DOI: 10.1016/j.seizure.2014.02.007. PMID: 24630806.
7. Beghi E., Carpio A., Forsgren L. et al. Recommendation for a definition of acute symptomatic seizure. *Epilepsia*

2010; 51: 671-675. DOI: 10.1111/j.1528-x. PMID: 19732133.
8. Baratova M. S., Atayeva M. A. The estimation of heart rhythm disturbances at the left atrial stunding on early stages of remodeling left ventricular //World medicine journal. – 2020. – №. 1. – С. 1.
9. Baratova M. S. et al. Periodontal diseases in military age persons and arterial hypertension //Asian Journal of Multidimensional Research (AJMR). – 2020. – Т. 9. – №. 4. – С. 111-113.
10. Akhmedov A. T. et al. Congenital heart defects in children and comorbidity //E-Conference Globe. – 2021. – С. 102-102.
11. Ахмедов А. Т. Особенности иммунной системы при врожденных пороков сердца //european journal of modern medicine and practice. – 2022. – Т. 2. – №. 3. – С. 35-40.
12. Evtushenko S.K., Simonyan V.A., Kotsenko Yu.I. Functioning open oval window and its connection with paradoxical cerebral embolism, migraine attacks and transient amnesia // Aktualt nutrition cardiuneurology: Materials of the conference. - Kshv, 2011 .-- S. 16-22.
13. Mironenko T.V., Sorokin Yu.N., Bakhtoyarov P.D. Selected issues of angioneurology. - Lugansk: LLC "Virtual Reality", 2008. - 276 p.