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MENTAL DISORDERS IN CEREBRAL PALSY

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Abstract. The article covers in detail modern approaches to complex diagnostics and correction of mental development disorders in children with cerebral palsy (CP). It is shown that mental disorders in CP are diverse and are represented by cognitive disorders (impaired perception, memory, attention, visual-motor coordination, intelligence and speech), residual organic borderline disorders (cerebrosthenic, neurosis-like, psychopathic syndromes), as well as pathological personality development (character accentuation, mental infantilism). Diagnostics of mental disorders in CP is a complex task due to their diverse combination with motor, speech and sensory pathology, which requires an integrated approach. The article describes differential diagnostic criteria for distinguishing between mental retardation and intellectual disability in cerebral palsy, and also covers in detail modern trends in comprehensive rehabilitation (including medical, social, psychological and pedagogical correction) of cognitive, emotional and behavioral disorders in patients with cerebral palsy.

Key words: cerebral palsy, mental disorders, cognitive disorders, mental retardation, mental retardation, neurosis-like syndrome**.**

Cerebral palsy (CP) is a polyetiological disease of the central nervous system that develops as a result of damage that occurred in the perinatal or early postnatal periods. At the same time, the effect of damaging factors on the developing brain determines the diversity of combinations of motor and sensory disorders and underlies mental dysfunction, which must be taken into account when substantiating restorative treatment and social rehabilitation of patients [1, 2, 20]. The incidence of cerebral palsy in the world is detected within 1.8-5, on average 2.5 per 1000 children [1,2,3,5,7,9,11]. The characteristics of mental development disorders in cerebral palsy depend on the etiological factors, the period of ontogenesis at the time of their action, localization, the degree of severity of brain damage, as well as the socio-pedagogical conditions in which the sick child lives [1, 6, 8, 10,12,14]. In children with cerebral palsy, residual organic cerebral insufficiency of the brain can be the basis for the development of both cognitive pathology and psychogenic reactions, pathological formation of personality due to the impact of unfavorable psychosocial influences caused by disability

[2,13,15]. Most researchers emphasize that the disharmonious mental development of children with cerebral palsy is determined primarily by disorders of the motor-kinesthetic analyzer, vision and hearing, as well as the characteristics of life and upbringing [4,19, 20].

Mental disorders in cerebral palsy are represented by: cognitive disorders (disorders of perception, memory, attention, visual-motor coordination, praxis, intelligence and speech); residual organic borderline disorders: cerebrosthenic, neurosis-like and psychopathic syndromes; various variants of pathological personality formation: mental infantilism, character accentuations [3, 16,17,18]. For a comprehensive study of cognitive functions, laboratory and instrumental methods are also used: functional MRI, the method of long-latency cognitive evoked potentials, computer tachistoscopy (to study visual perception, functional visual asymmetry of the brain), test computer systems ("Rhythmo-, Mnemo-, Bina test", "Psychomat", "Spike-Children"), determination of the level of autoantibodies to the nerve growth factor in the blood serum (as an early potential molecular predictor of mental development disorders), Eyetracing systems (based on the analysis of eye and head movements, which allows studying visual perception and visual-motor coordination) [1, 20]. Early comprehensive diagnosis of mental disorders, their timely comprehensive correction allow to significantly compensate for cognitive and emotional disorders, prevent pathological formation of personality, significantly reduce the degree of disability, which contributes to more effective social integration and successful social adaptation of patients with cerebral palsy.

The aim was to study autoimmune parameters in children with cerebral palsy.

Materials and methods. In the blood serum samples of all observed patients with cerebral palsy (n=110), as well as in the blood samples of the control group (n=30), the serum immunoreactivity of natural neurotropic autoantibodies in the blood serum was quantitatively determined using the ELI-N-Test reagent kit (Immunculus LLC). The kit is used to determine IgG autoantibodies that interact with antigens of neurons (NF200 protein), glial cells (GFAP), nerve fibers (MBP), Ca-dependent protein (S100), voltage-dependent Ca channel, β-endorphin and neurotransmitter receptors (cholinergic receptors, GABA receptors, glutamate NMDA and AMPA receptors, dopamine receptors, serotonin receptors, m-opiate receptors). The content of neurotropic autoantibodies (NAAT) was determined according to the method of A.B. Poletaev using the standard solid-phase enzyme immunoassay ELI-N-Test and the test kit of the same name from MIC Immunculus (Russia). The level of serum concentration of e-AT for each neuroantigen was expressed in arbitrary units (i.e., percentage deviation from the standard serum IR).

Results and discussion. The data obtained during the study indicate multiple immunopathological processes in children with cerebral palsy, involving both axons and myelin, as well as astrocytic glia and neurotransmitter regulation systems.

Demyelination and axonopathy are confirmed by the simultaneous increase in auto AT to NF 200 and MBP, which correlates with the severity of motor deficit in children with cerebral palsy. Reactive astrocytosis (increased autoantibodies to GFAP and S100 proteins) indicates chronic neuroinflammation and impaired blood-brain barrier permeability. Dysregulation of Ca^{2+}-dependent channels and neurotransmitter imbalance form the prerequisites for epileptiform activity, spasticity and cognitive impairment typical of cerebral palsy.

The combined increase in NF 200 and MBP titers indicates the presence of associated degenerative inflammatory changes in axons and their myelin sheaths.

β-endorphin protein is an endogenous opioid, a modulator of pain and stress response. Excessive formation of AT (6.6% vs. 0% in the control group), which can lead to dysregulation of antinociceptive pathways and increased chronic pain in patients

Also, undifferentiated determination of the content of e-AT in the blood serum was carried out for various isoforms of the corresponding receptors (GABA receptors, glutamate NMDA and AMPA receptors, dopamine receptors, serotonin receptors and choline receptors). Increased individual levels of serum immunoreactivity of various e-AT to neurotransmitter receptors were recorded, reflecting the state of neurotransmitter systems.

Different episodes of a decrease in auto AT (especially to regulatory neuropeptides) in some patients indicate more long-standing or severe neurodestructive processes and depletion of the humoral response. An increase in the level of e-AT to the ligand-binding sites of neurotransmitter receptors indicates corresponding changes in the neuronal system. In particular, Hol-P is involved in the regulation of the body's vegetative function, as well as in the regulation of cognitive function and memory.

Early comprehensive diagnostics of mental disorders, their timely complex correction allow to significantly compensate for cognitive and emotional disorders, prevent pathological formation of personality, significantly reduce the degree of disability, which contributes to more effective social integration and successful social adaptation of patients with cerebral palsy.

Conclusions. Thus, the obtained data demonstrate that most children with cerebral palsy develop a complex of autoimmune reactions affecting the structural proteins of neurons, myelin and astrocytes, as well as key elements of neurotransmitter and ion channel systems. Such an immune profile confirms the multifactorial nature of the

pathogenesis of cerebral palsy and emphasizes the need for complex therapy, including immunomodulation, neuroprotection and restoration of neuroglial-vascular interaction.

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