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COGNITIVE IMPAIRMENT IN ALCOHOLICS, TOXIC BRAIN DAMAGE

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Introduction. Cognitive impairments accompany numerous diseases of the brain. In diseases with a predominant lesion of the subcortical basal ganglia (toxic brain lesions, vascular cerebral insufficiency), subcortical-frontal cognitive impairments come to the fore in the clinical picture of cognitive disorders in the form of changes in planning and switching activities, decreased reaction speed and mental performance, impulsive behavior. Such disorders are usually accompanied by symptoms of depression and neurological disorders in the form of the revival of primitive reflexes, oligobradykinesia and frontal dysbasia [1, 2]. There are mild, moderate and severe cognitive impairments. Historically, the problem of cognitive disorders has been studied primarily in the context of dementia, which means the most severe cognitive impairment, leading to maladaptation in everyday life. Subsequently, much attention was paid to less pronounced disorders [3].

Keywords: cognitive disorders, brain disorders, vascular cerebral insufficiency, depression, neurological disorders.

The purpose of this study is to identify the features of cognitive deficits in alcoholic toxic encephalopathies.

Material and methods: 66 patients were examined. The 1st group consisted of 30 men with chronic alcoholism (average age -47.5± 6.6 years), the average duration of alcoholism -16.7±2.1 years. The control group consisted of 30 conditionally healthy men of representative age (average —47.2±4.7 years) and total work experience (14.2± 1.2 years) who do not abuse alcohol.

In order to identify the features of cognitive impairment, a neuropsychological study was used, including a complex neuropsychological system according to A.R. Luria.; the state of memory was assessed ("fourth extra" tests, "broken window", "performing triple counting", "performing simple counting operations", "selecting opposites", "10 words", "memorizing groups of pictures when played three times"), praxis (samples of "fist-rib-palm", Head, Ozeretsky), gnosis (recognition of crossed-out, superimposed images, non-speech noises and familiar melodies, showing a given finger based on a sample and name) and speech (tests for understanding logical and grammatical constructions, ordinal score from 1 to 10, enumeration of days of the week, months, completion of well-known proverbs, repetition of sounds, series of sounds, words and phrases). MMSE and FAB tests were used to diagnose moderate cognitive disorders, lesions of the frontal lobes or subcortical cerebral structures.

The severity of emotional disorders was studied using scales for assessing the asthenic state, personal and reactive anxiety, the level of neuroticism and psychopathization, and Tsung's depression. Computer electroencephalography (EEG) was performed with the determination of

cognitive EP according to the standard The method is based on a computer multifunctional complex for the study of EEG and VP Neuron Spectrum-4.

For statistical processing of the research results, the integrated system for complex statistical analysis and data processing Statistica 6.0 was used. The normality of the distribution of quantitative indicators was checked using the Shapiro—Wilk criterion. The Mann test was used to compare quantitative characteristics.—According to Whitney, the differences were considered statistically significant at $p<0.05$. The results were presented as the median and interquartile range (25th and 75th percentiles).

Results and discussion. In group 1, alcoholic encephalopathy manifested itself in the form of scattered neurological symptoms, psychovegetative and cognitive disorders, sleep—wake cycle disorders. Asthenic symptoms manifested themselves in the form of lethargy, fatigue, fatigue, rapid fatigue, a pessimistic mood with a paradoxical emotional reaction, irritable weakness, decreased performance, increased drowsiness, lack of satisfaction from sleep and a feeling of weakness, heaviness in the head in the morning. On the background. In 20% of patients with asthenic syndrome, neurological examination revealed trembling of the fingers, head, and eyelids; in 16.7%— impaired coordination of movements. Sleep disorders and decreased appetite were reported in 63.3% of patients. In addition to drowsiness, sleep disorders manifested themselves in the form of sleep disorders, pathologically early awakening. Sleep disorders were accompanied by nightmarish dreams, feelings of falling, suffocation, and fear. Vegetative disorders have been observed in 83.3% of patients in this group had cognitive disorders in 90.0%, and emotional—volitional disorders in 100%. Patients complained of a feeling of "tingling", "crawling goosebumps", numbness in the distal extremities, and a neurological examination revealed a polyneuropathic type of sensitivity disorders. Polyneuropathy was diagnosed in 63.3% of cases.

The study showed that the average group indicators of the mnestic sphere and the sphere of attention in patients of the 1st and 2nd groups significantly differed ($p<0.05$) from the indicators in the control.

Conclusion. Thus, when analyzing the identified disorders in patients with toxic encephalopathy, a general pathological stereotype of development can be identified. It has been established that the leading clinical manifestation of toxic encephalopathy is an organic personality disorder with cognitive impairments of varying severity and asthenic syndrome. Cognitive impairments in toxic encephalopathies have been confirmed decreased memory capacity, attention, associative-logical thinking, and changes in cognitive (P300) brain activity. Psychoemotional disorders observed in encephalopathy of any etiology are inextricably linked with cognitive disorders.

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