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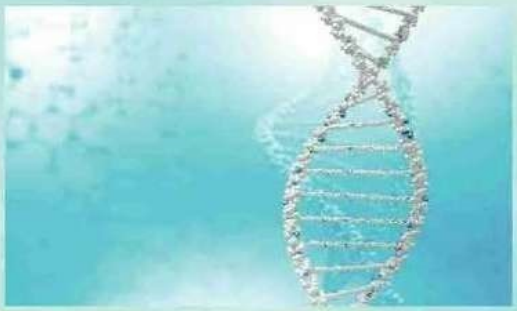

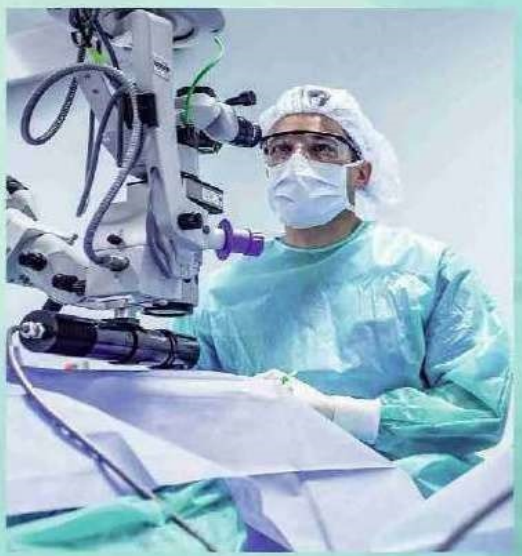

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NEUROCOGNITIVE ASPECTS OF PARANOID SCHIZOPHRENIA WITH CEREBRAL HEMODYNAMIC DISORDERS

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✓ ***Resume***

The article presents literature data and analysis of our own research on the study of neurocognitive aspects of paranoid schizophrenia with cerebral hemodynamic disorders. The data obtained in the work on impaired and preserved links of cognitive functions can be used for the optimal choice of rehabilitation measures, as well as for more accurate prediction of the expected effectiveness of complex therapy and the prospects for social adaptation of patients, depending on the presence of hemodynamic disorders.

Keywords: paranoid schizophrenia, neurocognitive profile, cerebral hemodynamic disorders

НЕЙРОКОГНИТИВНЫЕ АСПЕКТЫ ПАРАНОИДНОЙ ШИЗОФРЕНИИ С ЦЕРЕБРАЛЬНО-ГЕМОДИНАМИЧЕСКИМИ НАРУШЕНИЯМИ

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✓ ***Резюме***

В статье представлены литературные данные и анализ собственных исследований по изучению нейрокогнитивных аспектов параноидной шизофрении с церебрально-гемодинамическими нарушениями. Полученные в работе данные о нарушенных и сохранных звеньях когнитивных функций могут использоваться для оптимального выбора реабилитационных мероприятий, а также для более точного прогнозирования ожидаемой эффективности комплексной терапии и перспектив социальной адаптации пациентов в зависимости от наличия гемодинамических нарушений.

Ключевые слова: параноидная шизофрения, нейрокогнитивный профиль, церебрально-гемодинамические расстройства

ЦЕРЕБРАЛ-ГЕМОДИНАМИК БУЗИЛИШЛАРИ БИЛАН КЕЧАДИГАН ПАРАНОИД ШИЗОФРЕНИЯНИНГ НЕЙРОКОГНИТИВ АСОСЛАРИ

Абдуллаева В.К., Ирмухамедов Т.Б.

Тошкент педиатрия тиббиёт институти

✓ ***Резюме***

Мақолада церебрал гемодинамик бузилишлари билан кечадиган параноид шизофрениянинг нейрокогнитив асосларини ўрганиш бўйича адабиёт маълумотлари ва ўз тадқиқотларимиз таҳлили келтирилган. Когнитив функцияларнинг бузилган ва сақланиб қолган алоқалари бўйича ишда олинган маълумотлар реабилитация чораларини оптимал танлаш, шунингдек комплекс терапиянинг кутилаётган самарадорлигини ва беморларнинг ижтимоий мослашуви истиқболларини аниқроқ башорат қилиш учун ишлатилиши мумкин.

Калит сўзлар: параноид шизофрения, нейрокогнитив профил, церебрал гемодинамик бузилишлари

Relevance

Currently, there has been an increased interest in psychiatry in the neurobiological basis of schizophrenia and cognitive functioning. Neurocognitive deficit in schizophrenia is considered as a "third group of symptoms" along with positive and negative disorders [1, 3, 4]. It is believed that neurocognitive deficit largely determines the social and therapeutic prognosis of the disease, and also affects the formation of other psychopathological symptoms [8, 9]. Cognitive decline in patients with schizophrenia precedes the development of mental illness and is a genuinal disorder in the course of information processes [11, 13]. In recent years, there has been a tendency towards an increase in the proportion of young patients among patients with schizophrenia, a course with frequent exacerbations, increasing personality changes and a high level of disability [10]. The role of structural deficiency of the CNS in the development of a number of psychotic, especially hallucinatory, disorders in patients with schizophrenia has been proven [5, 7]. In such cases, in the affected organ, the likelihood of impaired vascular tone is increased. Vascular dystonia is caused by both structural deficiency in the affected organ and clinically "silent" structural abnormalities in other organs and systems, as well as increased "sensitivity" to psycho-traumatic factors, which was established mainly in early childhood [6]. A number of studies have made it possible to establish the predominant topic of cognitive deficit, which includes the interest of the prefrontal regions. Dysfunction of these brain structures forms the primary nature of disorders in the form of a decrease in control, programming and regulation of activity, as well as activation effects from the deep structures of the brain. Violation of the systemic mechanisms of interaction of subcortical formations with the frontal and temporal cortical zones with an emphasis on left-sided laterality is a genetic risk factor in schizophrenia [12]. Negative symptoms were significantly correlated with cognitive decline, behavioral disturbances, and neurological signs, including involuntary motor disturbances, but were not associated with previous therapy or positive symptoms. Positive symptoms did not correlate with any of the listed signs separately. All this led to a new paradigm shift in understanding the pathogenesis of schizophrenia, in which a significant role was attributed to neurocognitive deficit [14]. Currently, manifestations of neurocognitive deficits are considered as a key group of symptoms in schizophrenia, responsible, in particular, for impaired social functioning of patients.

Based on the foregoing, the **aim of the study** was to study the neurocognitive aspects of paranoid schizophrenia with cerebral hemodynamic disorders.

Material and methods

The study was conducted on the basis of the clinical psychiatric hospital in Tashkent. The study used data from clinical-psychopathological, pathopsychological (PANSS scale, Montreal Cognitive Assessment Scale (MoCA-test)) and neurophysiological studies of patients with paranoid schizophrenia, as well as anamnestic data, results of additional examination methods, and conclusions of a neurologist. A total of 108 patients with paranoid schizophrenia aged 25-50 years were included in the study. We formed two groups of patients: the main group consisted of 55 patients with a diagnosis of 55 patients with a diagnosis of F20 Paranoid schizophrenia with cerebral hemodynamic disorders and a comparison group - 53 patients with paranoid schizophrenia without comorbid cerebral pathology. Clinical verification of the diagnosis of F20 Paranoid schizophrenia was carried out using data from inpatient examination and observation, archival medical records. Selection criteria: presence of a clinically established diagnosis in accordance with the ICD-10 diagnostic criteria; the absence at the time of the examination of acute psychotic symptoms, pronounced intellectual-mnestic decline and signs of neuroleptic syndrome; absence of polymorphic symptoms; informed voluntary consent to participate in the study.

Results and discussion

At the first stage of the study, the patients underwent a transcranial Doppler study of cerebral hemodynamics using the duplex scanning method. In all studied cases, the slowdown in peak systolic and end-diastolic blood flow velocity was bilateral and symmetrical. In patients of the main group, all the studied parameters were significantly changed, in patients of the comparison group, the velocity characteristics of cerebral blood flow in the anterior, middle, posterior cerebral arteries were significantly slowed down, and the indices of peripheral vascular resistance, although slightly increased,

were not statistically significant. At the same time, there are statistically significant differences between the peak systolic blood flow velocity (the most significant parameter) in the anterior and middle cerebral arteries, as well as the index of peripheral vascular resistance in the middle cerebral artery in the examined patients. In cases where there were signs of early cerebral insufficiency in the premorbid period of patients with paranoid schizophrenia, a pronounced modification of the clinical picture of the endogenous disease was noted. One of the main clinical manifestations of the endogenous process in these patients was verbal hallucinosis, which had features of organic psychosis. Common to all manifestations of the endogenous process that develops in patients with cerebral insufficiency were extraordinary brightness, intensity, pronounced nature of painful experiences. Comparing the velocity parameters of cerebral blood flow in patients with paranoid schizophrenia, in whose anamnesis there were indications of biological hazards that could lead to cerebral dystonia, and patients without indications of the presence of such factors, the pulse systolic (57.2 ± 22.7 and 59 .1±16.8; $p<0.005$), end diastolic (38.1±13.7 and 32.5+11.8; $p<0.0045$) blood flow velocity and peripheral vascular resistance index (0.53±0 0.08 and 0.58±0.11; $p<0.005$) in the middle cerebral artery. Considering that structural and functional disorders of the brain are detected already at the initial stage of the course of schizophrenia and further progress as the course of the disease and the addition of concomitant pathology [2], in the studied patients, we studied the presence of somatic diseases that could affect the clinic and the course of schizophrenic process. Most often (45 people - 41.7%), diseases of the digestive system were noted: chronic hepatitis, cholecystitis, gastritis, colitis. In second place (26 people - 24.1%) were diseases of the cardiovascular system. Kidney diseases (urolithiasis and chronic pyelonephritis) were detected in 11 cases (10.2%). 12 patients (11.1%) suffered from respiratory diseases. Type II diabetes mellitus was detected only in 2 patients (1.8%). In the overwhelming majority of cases, the diagnosis of a concomitant somatic disease was first established in a psychiatric hospital during hospitalization for exacerbation of psychotic symptoms. Concomitant somatic pathology changed the clinical picture of paranoid schizophrenia. So, patients with diseases of the digestive system practically did not show any complaints characteristic of this pathology. In patients with schizophrenia, the threshold of pain sensitivity is increased, so these diseases proceeded in them atypically and without pain accompaniment. Patients complained of unusual, unpleasant sensations in the stomach and intestines, claimed that they heard voices reporting the decay of organs, their use for transplantation. Such psychopathological symptoms were often accompanied by aggressiveness, malice, indignation towards imaginary offenders. In the group of patients with paranoid schizophrenia with concomitant hypertension, the index of peripheral vascular resistance in the anterior cerebral artery also significantly differed from that in somatically healthy patients (0.58±0.11 and 0.63±0.1; $p<0.05$).

The study analyzed differences in the performance of cognitive tests between groups of patients. Thus, according to the MoCA test, visual-motor functions and visual memory are significantly more preserved in patients of the comparison group. The results of the study showed that verbal associative productivity and semantic memory reserve are significantly higher in patients without concomitant pathology. The nature and speed of execution, the number of points in the methods indicate that executive functions and control over actions are also more preserved in patients of the comparison group. Features of the pace and level of mental activity when performing simple mental work, indicators of concentration, switching, stability of attention and volume are higher in patients of the comparison group (Schulte tables). Patients with paranoid schizophrenia with concomitant cerebral pathology require more preparation for the main work than patients in the comparison group (Schulte tables). The operational functions of memory, the transition of traces to long-term memory are more preserved in this category of patients (subtest "Repetition of numbers", memorization test of 5 words). Constructive thinking, spatial analysis and synthesis, verbal abstract thinking are also more preserved in patients without concomitant cerebral pathology.

Conclusions

Thus, characterizing the neurocognitive profile of the examined patients, it can be determined that patients of the main group are inferior to patients of the comparison group in visual perception, hand-eye coordination, skill formation rate, mental stability and attention switching, constructive praxis, decreased executive functions, visual memory and reserve semantic memory. Due to a pronounced neurocognitive deficit, patients of the main group experience difficulties in daily activities to a greater

extent, they are less successful in overcoming life's difficulties, withstand internal and external stresses. The data obtained in the work on impaired and preserved links of cognitive functions can be used for the optimal choice of rehabilitation measures, as well as for more accurate prediction of the expected effectiveness of complex therapy and the prospects for social adaptation of patients, depending on the presence of hemodynamic disorders.

LIST OF REFERENCES:

1. Brugger S.P., Howes O.D. Heterogeneity and Homogeneity of Regional Brain Structure in Schizophrenia: A Meta-analysis. JAMA Psychiatry. 2017; 74(11):1104-1111. https://doi.org/10.1001/jamapsychiatry.2017.
2. Ceaser A.E., Barch D.M. Striatal Activity is Associated with Deficits of Cognitive Control and Aberrant Salience for Patients with Schizophrenia. Frontiers in Human Neuroscience. 2016;9:687. https://doi.org/10.3389/fnhum.2015.00687
3. Dietsche B., Kircher T., Falkenberg I. Structural brain changes in schizophrenia at different stages of the illness: A selective review of longitudinal magnetic resonance imaging studies. Australian and New Zealand Journal of Psychiatry. 2017;51(5):500-508. https://doi.org/10.1177/0004867417699473
4. Dorofeikova M., Neznanov N., Petrova N. Cognitive deficit in patients with paranoid schizophrenia: Its clinical and laboratory correlates. Psychiatry Research. 2018;262:542-548. https://doi.org/10.1016/j.psychres.2017.09.041
5. Green M.F., Harvey P.D. Cognition in schizophrenia: Past, present, and future. Schizophrenia Research: Cognition. 2014;1(1):1-9. https://doi.org/10.1016/j.scog.2014.02.001
6. Herold C.J., Essig M., Schröder J. Neurological soft signs (NSS) and brain morphology in patients with chronic schizophrenia and healthy controls PLoS ONE. 2020 Apr 22; 15(4): e0231669
7. Katsel P., Roussos P., Pletnikov M., Haroutunian V. Microvascular anomaly conditions in psychiatric disease. Schizophrenia - angiogenesis connection. Neurosci Biobehav Rev. 2017;77:327- 339. doi:10.1016/j.neubiorev.2017.04.003
8. Kong L., Herold C.J., Lässer M.M., Schmid L.A., Hirjak D., Thomann P.A. et al. Association of cortical thickness and neurological soft signs in patients with chronic schizophrenia and healthy controls. // Neuropsychobiology. 2015;71(4):225–33.
9. Medical comorbidity in schizophrenia Text. / D.V. Jeste, J.A. Gladsjo, L.A. Lindamer, J.P. Lacro // Schizophr. Bull. 2013. — Vol. 3. — P. 413-430.
10. Najjar S., Pearlman D.M. Neuroinflammation and white matter pathology in schizophrenia: systematic review. // Schizophr Res. 2015 Jan; 161(1):102-12.
11. Petrova N.N., Dorofeykova M.V., Voinkova E.E. (2016). Cognitive impairment in patients with schizophrenia at different stages of the disease. / Neurology and psychiatry. c.c. Korsakova, 116(4), 10-15.
12. Rosca E.C., Cornea A., Simu M. Montreal Cognitive Assessment for evaluating the cognitive impairment in patients with schizophrenia: A systematic review. General Hospital Psychiatry. 2020;65:64-73. https://doi.org/10.1016/j.genhosppsych.2020.05.011
13. Temur B., Irmuhamedov., Vasila K. Abdullaeva, Aleksey V. Rogov, Anastasiya A. Matveyeva, Kamola B. Sultonova, Dilorom S. Abbasova. (2021). Clinical and psychopatological characteristics of vascular diseases of brain in patients with paranoid schizophrenia. Annals of the Romanian Society for Cell Biology, 13080–13085. // Retrieved from https://www.annalsofrscb.ro/index.php/journal/article/view/4313
14. Vasila K. Abdullaeva, Temur B. Irmuhamedov, Kamola B. Sultanova, Dilorom S. Abbasova, Anastasiya A. Matveeva, Aleksey V. Rogov. Premorbid features of patients with paranoid schizophrenia with concomitant cerebral hemodynamic disorders. // European Journal of Molecular & Clinical Medicine, Volume 7, Issue 2, 2020, pp. 2320-2327.

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