



The role of the BDNF level in the formation of cognitive violations in people who have experienced COVID-19

Gafurov Baxtiyor Gafurovich and Mamadjonova Tursunoy Toxir qizi

Professor of center for the Development of Professional Qualifications of medical Workers, Uzbekistan.

Researcher of center for the development of professional qualifications of medical workers, Uzbekistan.

E-mail: cool.bahtiyar@yandex.com

E-mail: mamadjonovatursunoy9@gmail.com

ABSTRACT

We examined 80 patients who had undergone COVID-19. The main group consisted of 25 patients with cognitive impairment who underwent coronavirus disease, which was confirmed by the presence of positive tests and antibodies for COVID-19, the comparison group consisted of 34 patients without cognitive impairment who had a coronavirus disease, which was confirmed by the presence of positive tests and antibodies for COVID-19. The control group consisted of 21 patients of the same age without cognitive disorders. To study clinical, neurological, neuropsychological parameters and the quantitative content of brain-derived neurotrophic factor (BDNF) in cognitive disorders in patients who underwent COVID-19.

Keywords: COVID-19, BDNF, cognitive function, MoCA, MMSE, Dementia.

Received 13.08.2023

Revised 03.10.2023

Accepted 21.10.2023

INTRODUCTION

At least one third of patients with COVID-19 in the acute and / or residual phase have neurological disorders. Most often, asthenic, cognitive impairments, confusion, strokes, peripheral neuropathies, anosmia, dysgeusia, and pain syndromes are recorded. The most likely mechanisms of damage to the nervous system in COVID-19 are pathological immune responses, cerebrovascular diseases due to coronavirus endothelitis, and hypoxic brain injury; the direct neurotropic effect of the virus is also discussed [2,6,7]. The ability of the SARS-CoV-2 virus to infect cells of the nervous system carries potential risks of long-term neurological complications. The long COVID condition, which is characterized by dysfunction not only of the lungs due to pulmonary interstitial fibrosis, but affects all levels of the nervous system, can have a serious impact on quality of life. It has been suggested that neuronal damage caused by SARS-CoV-2 may also be a driving force behind chronic degenerative diseases of the nervous system. Regardless of direct or indirect exposure to the virus, damage to the central and peripheral nervous system due to COVID-19 may become irreversible.

Damage to the nervous system confirms the neurotropism of the COVID19 virus and the presence of neuroinflammatory syndromes [4,5].

Since the beginning of the COVID-19 pandemic, there have been reports of a frequent association of the new coronavirus infection with neurological disorders.

Chinese doctors were the first to show a significant prevalence of severe and persistent headache that did not correspond to the severity of general intoxication, frequent episodes of delirious confusion, also not corresponding to the severity of general intoxication, and a decrease in smell and taste [1,4].

There is now sample evidence that mild to moderate cognitive impairment may be the most common neurological consequence of novel coronavirus infection. British neuropsychologists conducted online testing of more than 84,000 patients over 16 years of age who did not have any cognitive difficulties before the coronavirus infection. The results of cognitive tests significantly differed from age standards both in terms of integrative indicators, and especially in the areas of memory and attention; at the same time, the degree of deviation from the norm depended on the severity of the coronavirus infection [3,4].

MATERIAL AND METHODS

All patients underwent a clinical and neurological examination with a detailed history taking. To assess the level of cognitive deficit, the MMSE Test (Mini-Mental State Examination), a brief examination of cognitive function, and the Montreal Cognitive Scale (MoCA, Montreal Cognitive Assessment) were used. The studies were carried out in a separate room in a confidential and calm atmosphere. The responses were evaluated according to the following criteria: total score is 30. MMSE assessment criteria: 30 - maximum score, 27–25 - moderate cognitive impairment, 24 or less points - severe cognitive impairment (dementia). The Montreal Cognitive Assessment (MoCA, from the English. Montreal Cognitive Assessment) is a widely used screening for cognitive impairment, the questionnaire is a one-page test of 30 items, performed on average in 10 minutes. All patients underwent blood sampling for BDNF testing.

RESULT AND DISCUSSION

Of the complaints of patients who underwent COVID-19, psycho-emotional disorders prevailed, signs of asthenia in the form of weakness and decreased performance in 20 patients of the main group (80%) and 18 patients of the comparison group (53%), fatigue in 17 patients of the main group (68%) and 12 patients of the comparison group (35%), 14 (56%) patients of the main group and 9 (26%) patients of the comparison group complained of headache, dizziness - 15 patients of the main group (60%) and 10 (29%) patients of the comparison group, night sleep disturbance in 16 patients (64 %) of the main and 12 (35%) comparison groups, excessive irritability and nervousness in behavior were noted by 19 patients (76%) of the main and 13 (38%) comparison groups who had had coronavirus disease. It should be noted that all patients of the main study group complained of a decrease in memory and attention.

Analysis of focal neurological symptoms showed: central paresis of the seventh pair of cranial nerves was detected in 11 (44%) of the main and 3 (9%) comparison groups, central paresis of the XII pair of cranial nerves occurred in 5 (20%) of the examined patients of the main group, respectively. Reflexes of oral automatism occurred respectively in 6 (24%) of the main group, anisoreflexia was diagnosed in 18 (53%) patients of the main group, unsteadiness in the Romberg position in 16 (64%) patients of the main group. We present the results of experimental psychological studies - analysis of the cognitive sphere of patients who have undergone COVID-19. The neuropsychological study included the Mini Mental Status Assessment (MMSE) and the Montreal Cognitive Scale (MoCA). In the control group, the mental state indicators on the MMSE scale revealed the sum of points equal to 29.5 ± 0.1 , according to MoCA - 28.01 ± 0.1 , in the comparison group 28.4 ± 0.1 , according to MoCA - 28.04 ± 0.1 , which was close to normal (30 points) and the absence of cognitive impairment. In all patients of the main group, the sum of points on the MMSE scale was 23 ± 1.02 , according to MoCA - 17.6 ± 1.02 , which corresponds to more pronounced cognitive disorders prone to dementia.

In the control group in men and women, according to the MMSE and MOCA tests, moderate cognitive disorders were revealed. In both groups, patients over 40 years of age had severe cognitive impairment. The degree of moderate and severe cognitive impairment in general did not depend on the level of education in patients of both groups, both with secondary and higher education was identical.

Table1: Scoring severity of cognitive impairment in the examined groups

Groups	Patience with Covid-19		Control groups	
	MMSE	MOCA	MMSE	MOCA
Group as a whole (average)	$25,6 \pm 0,2$	$22,6 \pm 1,2$	$25,5 \pm 0,1$	$24,1 \pm 1,2$
sex:				
M	$24,6 \pm 1,8$	$21,8 \pm 1,1$	$26,8 \pm 3,8$	$25,4 \pm 1,2$
F	$26,2 \pm 1,4$	$23,2 \pm 0,4$	$25,8 \pm 1,7$	$25,2 \pm 2,4$
age: under 40 years above 40 years				
under 40 years	$26,4 \pm 2,1$	$22,9 \pm 2,3$	$26,4 \pm 1,6$	$24,4 \pm 1,1$
above 40 years	$24,4 \pm 1,7$	$21,3 \pm 4,1$	$24,4 \pm 3,8$	$24,8 \pm 3,4$

In order to study the relationship between cognitive impairment and anxiety, we divided patients who had Covid into two groups: those with high anxiety and those with low anxiety. In both groups, MMSE and MOCA tests were performed, as a result of which patients with high anxiety were found to have more pronounced cognitive impairments compared to those with low anxiety. In women in the group with high anxiety according to MMSE and MOCA, severe cognitive impairment was noted. Also, in patients with high anxiety older than 40 years, compared with patients with low anxiety, cognitive impairment was also noted.

It should be noted that in this group, cognitive impairment was noted before COVID-19 disease. Coronavirus disease contributed to an increase in cognitive deficits, which is consistent with other studies [4]. One of the factors contributing to the control of neuronal metabolism in oxygen deficiency in patients

with a history of coronavirus infection is a brain-derived neurotrophic factor (BDNF) that promotes neuroplasticity of brain cells, i.e. protects brain neurons from ischemic attacks and death.

Index	Control (N ^o 21)	Sick «CF -» (N ^o 34)	Sick «CF +» (N ^o 25)
BDNF	859.4±3.6	675.4±2.3	101.9±2.4

In our study, the level of brain-derived neurotrophic factor (BDNF) in the group of patients who underwent COVID-19 with cognitive impairment was 101.9±2.4, while in the group of patients who underwent COVID-19, without cognitive impairment - 675.4±2.3, while in the control group the indicator was 859.4±3.6, which confirms the decrease in neurotrophic factor in cognitive impairment.

CONCLUSIONS

Thus, a study of cognitive status and biochemical studies showed that cognitive deficits in patients who underwent COVID-19 accompanied by a decrease in the level of brain-derived neurotrophic factor (BDNF), which confirms the pathogenetic mechanisms of cognitive deficit in coronavirus infection.

REFERENCES

1. Mao L., Jin H., Wang M. et al. (2020). Neurologic manifestations of hospitalized patients with coronavirus disease 2019 in Wuhan, China. *JAMA Neurol.* Vol. 77. № 6. p. 683-690.
2. DosSantos MF, Devalle E, Aran V, et al. (2020). Neuromechanisms of SARS-CoV-2: A Review. *Front Neuroanat.*6;14:37. doi: 10.3389/fnana.2020.00037. eCollection 2020.
3. Bohmwald K, Galvez NMS, Rios M, Kalergis AM.(2018). Neurologic Alterations Due to Respiratory Virus Infections. *Front Cell Neurosci.*26;12:386. doi: 10.3389/fncel.2018.00386. eCollection 2018.
4. Gu J, Gong E, Zhang B, et al. (2005). Multiple organ infection and the pathogenesis of SARS. *J Exp Med.* ;202(3):415-24. doi: 10.1084/jem.20050828. Epub 2005 Jul 25.
5. Castren E, Vöikar V, Rantamäki T. (2007). Role of neurotrophic factors in depression. *Curr Opin Pharmacol.* 7(1):18–21. DOI: 10.1016/j.coph.2006.08.009
6. Kuipers SD, Dramham CR. (2006). Brain-derived neurotrophic factor mechanisms and function in adult synaptic plasticity: new insights and implications for therapy. *Curr Opin Drug Discov Devel.* 9(5):580–586.
7. Leibrock J, Lottspeich F, Hohn A, et al. (1989). Molecular cloning and expression of brain-derived neurotrophic factor. *Nature.* ;34(6238): 149–152. DOI: 10.1038/341149a0
8. Tool J. F. (2007). *Vascular diseases of the brain; management for doctors.* – M.: GEOTAR-Media; 590s.
9. N.N. Yakhno, V.V. Zakharov, A.B. Lokshina, N.N. Koberskaya, E.A. Mkhitaryan. M.(2010). *Dementia: a guide for doctors* /.: MED-press-inform; 272p.

CITATION OF THIS ARTICLE

Gafurov Baxtiyor Gafurovich and Mamadjonova Tursunoy Toxir qizi. The role of the BDNF level in the formation of cognitive violations in people who have experienced COVID-19. *Bull. Env. Pharmacol. Life Sci.*, Vol 12[11] October 2023: 32-34