

**NEUROPSYCHOLOGICAL STATE OF POST-COVID PATIENTS**

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**Abstract:** The purpose of this review is to summarize and analyze published literature data during the COVID-19 pandemic on possible pathogenetic mechanisms for the development of neuropsychiatric and cognitive impairments (CI) in patients with coronavirus infection. According to currently available data, SARS-CoV-2 can affect every organ in the body, leading to acute damage and long-term consequences, with the latter effects becoming apparent only recently.

**Keywords:** COVID-19, SARS-CoV-2, coronavirus, psychoneuroimmunology, cognitive impairment.

The mechanism of damage to the nervous system during viral infections is complex and continues to be studied. The polymorphism of the morphological and clinical manifestations of the viral disease itself is associated with a variety of pathogenetic mechanisms: the direct toxic effect of the virus, activation of other microorganisms and viruses, secondary vascular disorders, etc.

The assumption about the presence of the neurotropic potential of the SARS-CoV-2 virus is confirmed by the study of L. Mao et al., which shows that 78(36.4%) patients out of 214 patients with COVID-19 had clinical manifestations of damage to the nervous system [1]. Disorder of respiratory function in COVID-19 is considered by a number of authors to be the result of a violation of the central regulation of breathing due to multifocal symmetrical lesions of the brain stem [2, 3].

It should be noted that the pandemic is a significant psychological stressor in addition to its enormous impact on all aspects of the lives of individuals and society as a whole. Fear of illness and uncertainty about the future provokes anxiety and stress disorders, and some experts rightly call for the creation and sharing reliable mental health screening and treatment programs to the general public and health care providers. However, beyond pandemic-related psychological distress, the direct impact of the virus itself (several acute respiratory syndrome coronaviruses; SARS-CoV-2) and the following impact of the immunological response on the central nervous system and associated outcomes are unknown [1, 2]. Prospective neuropsychiatric monitoring of individuals, exposed to SARS-CoV-2 at different periods of life, and also assessment of their neuroimmune status is necessary for complete understanding the long-term impact of COVID-19 and creating a framework for integration of psychoneuroimmunology into epidemiological research pandemics [6]. COVID-19 infection through immune activation inflammatory processes and cytokine storm can lead to manifestations of neuropsychiatric symptoms, also to increase of severity of psychiatric symptoms in those infected with COVID-19 persons with pre-existing mental disorders. The given assumption may expand the understanding of the importance of even mild chronic inflammation and cytokine storm in psychoneuroimmunology. Accordingly, patients who have had COVID-19 from older age groups, taking into account cognitive comorbidity in are more susceptible to the progression of CI, so as on the background of age-

related changes the accumulation of the defect and decompensation are observed with the progression of a particular disease.

It is necessary the early identification (on admission) of patients with anxiety disorders and high levels of anxiety, as well as addictions (alcoholism, addiction to benzodiazepines, cannabinoid and opioid addiction) existing before admission [4]. After stay in intensive care and anoxic damage in patients anxiety, depression and CI develop. Need organization and execution neuropsychological rehabilitation, psychological consultations, psychological support, cognitive training [5]. In patients, survivors of COVID-19 often develop mental disorders: depression, anxiety disorders [6]. And depression, in turn, can aggravate existing CIs or be their cause in itself (so called pseudodementia) [7]. Therefore, a correction of emotional disorders leads to beneficial effect on cognitive abilities.

In the treatment of depression in people with CI, drugs with strong anticholinergic effects, such as tricyclic antidepressants, should be avoided, as they may worsen CI. On the contrary, modern antidepressants from the group of selective serotonin reuptake inhibitors (fluoxetine, paroxetine, fluvoxamine, etc.), vortioxetine have a positive effect on cognitive functions [7].

Thus, after the end of the acute period of viral infection, patients may experience an increase in cognitive impairment in all age groups, especially in the presence of comorbid cognitive impairment. Post-Covid neurological disorders lead to a significant decrease in the quality of daily life in a large number of patients, including patients of working age. The mechanisms of formation of changes in the nervous system during a new coronavirus infection are not fully understood.

### **Influence of psychological factors on the phenomenon of coronosomnia**

We conducted a study of 30 patients (77% of women and 23% of men in the age range of 18-37 years), who were asked to complete the diagnostic scales questionnaire after 10 days after the diagnosis of coronavirus infection. in the city of Andijan.

#### **Research methods:**

- short health assessment Questionnaire (Patient Health Questionnaire-2);
- шкала оценки симптомов Insomnia Severity Index (Insomnia Severity Index, ISI);

The results revealed that 48% of the participants had severe sleep disorders in the form of presomnic disorders. Insomnia symptoms were significantly higher in women (14.8; SD=4.28) than in men (10.18; SD=3.11),  $t(23.19)$  Those who answered "I do not know" when asked if they had contracted the coronavirus scored significantly higher on insomnia (14.25, SD =4.11),  $F(2.281) = 11.27$ ,  $p \leq 0.001$  than those who answered "yes". or not." The same was observed when answering the question whether someone close to them was infected with coronavirus (14.10; SD 4.23;  $F(2.213) = 11.21$ ;  $p \leq 0.001$ ). To predict the severity of insomnia symptoms, multiple linear regression was performed based on the significance of the positive correlation of independent variables. An important equation was found:  $F(4,2192) = 310.72$ ,  $p \leq 0.001$ ,  $R^2=0.383$ . Predicted insomnia during the COVID-19 pandemic is + 0.728 (tolerance to uncertainty) + 0.619 (covid-associated anxiety) + 0.471 (feeling lonely) + 1.338 (symptoms of changes in mental health).

**Discussion of the results.** We have shown that women are more likely to change their sleep patterns during a pandemic, and this finding is consistent with foreign data showing that women

are more likely to have stress-related disorders such as PTSD and anxiety spectrum disorders. Anxious ruminations that cause cognitive arousal, unpleasant physical reactions also affect presomnic disorders. This is consistent with data from [3] and may also explain the high scores on the insomnia rating scale for those who answered "I do not know" when asked whether they or someone close to them had contracted the virus, since this answer implies uncertainty. Anxiety about coronavirus infection has also been shown to be associated with insomnia. The presence of waiting anxiety causes cognitive arousal and therefore affects the ability to sleep. Subjective feelings of loneliness are also associated with symptoms of insomnia. There is a study by our foreign colleagues on the bidirectional relationship between loneliness and insomnia [7]). Loneliness can lead to increased feelings of vulnerability, hence cognitive and behavioral hyperarousal, anxious and shallow sleep. On the contrary, poor sleep increases the frustration associated with feelings of isolation and can interfere with contact with other people, for example, due to a disrupted sleep — wake schedule [6]. Our study found a link between depression symptoms and sleep. This phenomenon is well described in a number of studies, as insomnia is considered an important precedent for depression, as well as a predictor of relapse of a depressive episode.

#### **Literatures.**

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