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NEUROPSYCHIATRIC DISORDERS THAT DEVELOP IN A COMPLICATION OF COVID-19 TO ALCOHOLISM

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Abstract

The development of neuropsychiatric disorders in patients with Postcode syndrome alcoholism is a problem in which the combined effect of the virus on neurotropicity, systemic inflammation and stressful psychological factors is an important issue today. Polymorphism of the clinical consequences of SARS-CoV-2 infection causes difficulties in diagnosing and providing medical care to patients with covid-19. There is no single approach and generally accepted terminology in the treatment of many phenotypic manifestations of postkovide.

Keywords: alcoholism, COVID-19, SARS-CoV-2 infection, psychological factors

INTRODUCTION

Postcovid syndrome is regarded as a complication of COVID-19 associated with direct viral damage to the central nervous system and systemic disorders (primarily cardiovascular and respiratory), as well as stress factors [1, 3, 6]. Infection caused by SARS-CoV-2 is a systemic disease, therefore the pathogenesis of psychopathological symptoms appears to be multifactorial. Coronaviruses are able to induce psychopathological complications through direct exposure to the central nervous system or indirectly through an immune response [2, 4, 7]. Clinical, postmortem, biological, in vitro, and cell culture studies have demonstrated the potential neurotropy and ability of coronaviruses to cause neuronal damage [5, 8, 9]. A "cytokine storm" can lead to psychiatric symptoms through the process of neuroinflammation [10, 11, 12]. The probability of neuropsychiatric complications in the postcovid period is associated with an increased index of systemic immune inflammation. Among the inflammatory molecules, TNF-alpha, IL-6, IL-1 beta, interferon-gamma, granulocyte-macrophage colony stimulating factor are distinguished. Mast cells are activated and also support inflammation due to histamine, cytokines and chemokines [13]. Elevated levels of pro-inflammatory cytokines contribute to their penetration through the blood-brain barrier and activation of astrocytes and microglia. Microglial cells and astrocytes begin to synthesize IL-1

beta, the receptors for which are distributed in the hippocampus zone [14]. Activation of glial cells and astrocytes affects the exchange of neurotransmitters, growth factors involved in the process of differentiation and survival of neurons [15].

Stigmatization due to coronavirus infection, traumatic memories of the past illness, social isolation, deterioration of material well-being are powerful psychological triggers that influence the development of mental disorders and determine the psychopathological outcome [16, 17].

There is evidence that patients with comorbid diseases and immunosuppression are more susceptible to the development of cognitive impairment, anxiety and depression [18]. Respiratory symptoms of COVID-19 may cause the development of panic disorder [19]. The development of affective disorders in the postcovid period is partly explained by systemic inflammation due to COVID-19, when the hyperinflammation reaction in the acute phase continues into persistent chronic inflammation, which is a pathogenetic mechanism of mood disorders [20, 21].

Among the leading factors associated with depression, there is also a female gender, a previous psychiatric history [22].

An inverse correlation was found between the severity of depressive symptoms and the duration of hospitalization of patients [23]. Coronavirus seropositivity is associated with suicidal behavior within a year after acute illness [25]. An imbalance of central neurotransmitters (norepinephrine, epinephrine, serotonin) can also affect the development of post-corneal affective disorders [24, 26].

Women and patients with a previous diagnosis of depression or anxiety disorder have a greater risk of developing fatigue [27].

Endothelial dysfunction in the capillaries of the brain may be a probable physical basis for fatigue in the postcovid period [28]. The severity of asthenic syndrome does not correlate with the severity of the disease, but it is significantly higher in patients who have passed through the intensive care unit [27, 29].

The development of cognitive impairment against the background of COVID-19 is multifactorial and is not directly related to the severity of the disease. It is assumed that the SARS-CoV-2 virus is able to reduce the activity of the brain neurotrophic factor, increase the levels of markers of axoal damage, which indicates potential damage to the central nervous system during the acute phase of the disease. The pathogenetic mechanisms of cognitive impairment in COVID-19 include respiratory failure and hypoxia, cytokine storm, hemostasis disorders, damage to the endothelium of the microcirculatory bed, direct brain damage, possibly by the mechanism of molecular mimicry [31, 32]. Disorders of a number of cognitive functions (executive functions and attention) are accompanied by changes in glucose metabolism in the prefrontal, subcortical and insular regions of the brain [30].

COVID-19 increases the risk of impaired cognitive functioning regardless of the presence of previous cognitive impairments [33, 34]. Acute respiratory syndrome and associated hypoxia affect the deterioration of attention, verbal and executive functions [35]. In one of the studies, the severity of depressive symptoms was a predictor of cognitive impairment in patients who had a coronavirus infection [36].

Conclusion

The long-term negative impact of COVID-19 on public health is obvious. Currently, large-scale studies are underway to study the prevalence of depression, anxiety, delirium and post-traumatic stress disorder in patients with COVID-19 [49]. These patients have a number of long-term multisystem symptoms without proven organ damage and with normal physical and laboratory parameters, accompanied by a decrease in performance of varying degrees of severity, associated, among other things, with cognitive impairment, equally represented in all age groups.

Postcovid syndrome is a socially significant disease that requires dynamic monitoring of patients and the development of rehabilitation programs. A wide range of different patient examinations and an integrative interdisciplinary approach to treatment and rehabilitation care are needed, combining traditional pharmacotherapy, non-pharmacological methods of treatment, behavior and lifestyle changes. The development of a unified public health strategy aimed at combating the adverse long-term consequences of the pandemic is relevant.

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