NEUROLOGICAL DISORDERS IN COVID-19

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Many COVID-19 patients have neurological disorders such as headache, dizziness, nausea, vomiting, neck muscle tension, and impaired sense of smell and taste. Although in many cases such symptoms may be insignificant against the background of acute respiratory disorder, nevertheless, encephalitis, meningitis, cerebrovascular disorders, depression, and other mental disorders have been identified. Viral neuroinvasion also contributes to the development of exacerbations and progression of acquired (myasthenia gravis, multiple sclerosis, opticomyelitis, inflammatory, autoimmune, paraproteinemic chronic polyneuropathies), hereditary demyelinating, metabolic, neurodegenerative and neuromuscular diseases. The appearance of neurological symptoms is an indicator of a poor prognosis in the course of the disease.

Aim. The purpose of this review is to provide an overview of the mechanisms of development of neurological disorders in COVID-19

Materials and methods. Data analysis of literature and Internet sources.

Results and discussion. Nowadays, there are two main pathogenetic mechanisms leading to the development of various neurological disorders: direct invasion of the virus into the nervous tissue and a maladaptive inflammatory response. Direct invasion can be carried out through infection of endothelial cells of blood vessels, on which ACE_2 receptors are expressed in large quantities. As a result of infection of endothelial cells, the integrity of the blood-brain-barrier is disrupted and its permeability increases. As a result, the virus enters directly into the brain tissue and can attack those cells on which ACE_2 receptors are expressed. The second mechanism by which the virus enters the brain tissue is the dissemination of SARS-CoV-2 through the ethmoid bone and olfactory bulbs. The virus enters the central nervous system through the terminals of the olfactory nerves and, due to the retrograde axonal current, enters the bodies of neurons, and after replication attacks other neuronal cells. However, viral particles are not always found in the neuronal cells of virus-infected areas of the brain. In this regard, it can be assumed that brain damage may not occur due to direct invasion of the virus, but be a consequence of the so-called "cytokine storm", when inflammatory cytokines are released from infected neurons and an acute maladaptive inflammatory response develops. In addition, damage to the nervous system can occur due to hypoxia, sepsis, multi-organ damage, and a hyperinflammatory response of the immune system - the cytokine storm.

Conclusions. Thus, neurological disorders in COVID-19 are caused by hypoxemia, impaired homeostasis (critical encephalopathy), neurotropic action and neurovirulence SARS-CoV-2 (isolated cranial nerve damage, focal and diffuse CNS lesions), "cytokine storm", as well as mixed impact of these factors.