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Post-Covid Trigemenal Neuralgia

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Annotation: Trigeminal neuralgia is one of the pressing problems of modern medicine. Trigeminal neuralgia (TN) is characterized by severe pain and usually occurs in elderly and senile people. There are many reports of the development of acute polyneuropathy in patients with coronavirus infection. This article provides a review of the literature on modern concepts of post-Covid trigemenal neuralgia.

Keywords: neuralgia, trigeminal nerve, coronavirus, pain syndrome.

Relevance. Trigeminal neuralgia (facial or trigeminal neuralgia) is a disease of the peripheral nervous system characterized by short-term, intense and recurring pain in the area of innervation of one or more branches of the nerve. The prevalence in the population is estimated at 1 case per 15 thousand people (may be underestimated due to incorrect diagnosis). It most often occurs in people over 50 years of age, but can also occur in young people with multiple sclerosis. More common in women.

Infection caused by the coronavirus SARS-CoV-2 (COVID-19) is accompanied by numerous lesions of various organs and systems of the human body and often leads to death, especially in the presence of risk factors (old age, abdominal obesity, severe arterial hypertension, diabetes mellitus) . There are many reports of the development of acute polyneuropathy in patients with coronavirus infection. In an observational study, 8.9% of 214 patients with confirmed COVID-19 had peripheral nerve symptoms, including hypogeusia, hyposmia, hypoplasia, and neuralgia.

Neurologists and infectious disease specialists describe frequent damage to various parts of the peripheral nervous system with this viral disease due to the exacerbation and acute course of herpetic infections. Although coronavirus disease 2019 (COVID-19) primarily manifests as pulmonary symptoms, doctors have recognized extrapulmonary symptoms, including skin symptoms. Cutaneous manifestations in patients with COVID-19 include acroischemic, chilblain rashes, petechiae and purpura, vesicles, urticaria, and erythematous maculopapules. Herpes zoster (HZ) is caused by the varicella zoster virus. Patients at risk for HZ include the elderly and immunocompromised individuals. Although COVID-19 is known to affect the immune system and may increase the risk of HZ, many studies support a link between HZ and COVID-19. There are reports in the literature that SARS-CoV-2 may be a risk factor for Herpesviridae reactivation. Varicella zoster virus reactivation following COVID-19 vaccination: a systematic review of case reports."

Trigeminal neuralgia is a compression neuropathy with a specific paroxysmal manifestation of symptoms of irritation. Trigeminal neuralgia (classical neuralgia) is characterized by short-term attacks of a shooting or cutting nature. With trigeminal neuralgia, there are trigger zones on the skin of the face or in the mucous membrane of the mouth. Irritation of these areas (touch, cold wind, displacement of the skin) usually causes pain. Trigger zones appear during the period of exacerbation of the disease; in remission they subside. Often the trigger zone in patients is the teeth. When examining patients, pain is often noted at the exit site of the corresponding branch of the trigeminal nerve on the face. Sometimes there are areas of hyperesthesia in the area of the affected branch. The clinical picture may change as the disease progresses: in pauses between attacks, a feeling of dull pain and burning may persist, hyperesthesia is replaced by hypoesthesia and even anesthesia. However, signs of neuralgia persist (painful paroxysms and trigger zones).

The intensity and suddenness of the onset of pain, the repeated nature of attacks leads to physical and mental exhaustion, and give grounds to consider trigeminal neuralgia the most difficult disease to

tolerate (Yusupov A.K., 2024). The trigeminal nerve, the fifth pair of cranial nerves, is the largest of the 12 pairs of cranial nerves. It belongs to the nerves of a mixed type, including sensory and motor fibers. The trigeminal nerve originates in the cranial cavity, in the Gasserian ganglion. A sensitive nerve root departs from this node, which almost immediately divides into 3 branches. They come to the surface through natural openings in the bones of the skull and provide the face with sensitivity to touch, pain, heat and cold. I branch innervates the forehead, eyebrow and eye, II branch is responsible for the sensitivity of the cheek, nose, upper lip, upper jaw and palate, III covers the chin, lower lip, lower jaw, tongue and temple, usually attacks of pain begin from areas lying in areas of innervation of the II and III branches.

Coronaviruses have a pronounced tropism for the epithelium and affect the mucous membranes of the respiratory tract, conjunctiva, oral cavity, esophagus and stomach [10], the innervation of which is carried out by the terminal branches of the olfactory (I), trigeminal (V), facial (VII), glossopharyngeal (IX) and vagus (X) nerves. Identification of SARS-CoV-2 virus RNA plays a critical role in the diagnosis of COVID-19. RNA of the SARS-CoV-2 virus is detected in tear fluid, smears from the oropharynx and nasopharynx, saliva, sputum, bronchial lavages, cerebrospinal fluid, in samples of lung parenchyma and brain tissue during autopsies of those who died from COVID-19 [11, 12]. Damage to the peripheral nervous system in COVID-19 can be represented by multiple cranial neuropathy, polyneuropathy, neuropathies of individual cranial and peripheral nerves, which are often combined with each other [9]. The reasons for the development of neuropathies in COVID-19 remain unclear. In some cases, the SARS-CoV-2 virus appears to be directed against antigens of Schwann cells and myelin. In other cases, neuropathies may be caused by vasculitis of the small vasa nervorum, leading to nerve ischemia. Neuropathies in COVID-19 can manifest as multiple mononeuropathy, asymmetric polyneuropathy, and classical symmetric polyneuropathy [13, 14]. Cranial neuropathy can be isolated or occur as part of multiple mononeuropathy. Most often, the olfactory (I), facial (VII), glossopharyngeal (IX), optic (II) and vagus (X) nerves are involved [15].

As is known, the “key” that allows the SARS-CoV-2 virus to enter the cell is the receptor for the angiotensin-converting enzyme. This type of receptor is widely represented in the central nervous system and is present both on the membrane and in the cytoplasm of neurons and gliocytes, in the cortex and subcortical gray ganglia [5]. Therefore, theoretically, we can expect some patients with COVID-19 to develop viral encephalitis. Two possible routes of penetration of SARS-CoV-2 into the brain are discussed: from the bloodstream through the blood-brain barrier along with immunocompetent cells and through the nasal mucosa and olfactory nerve fibers [5]. However, in practice, the number of cases of proven viral encephalitis of coronavirus etiology is very small [6].

Structural and functional changes in various parts of the nervous system play a leading role in the formation of the clinical syndrome of trigeminal neuralgia. “Peripheral” hypotheses suggest initial damage to the peripheral parts of the nervous system, while “central” hypotheses emphasize the importance of intracerebral nuclear and conductive structures (1, 5). The results of surgical treatment of trigeminal neuralgia showed that the main factor leading to the development of neuralgic pain syndrome is the initial damage to the peripheral part of the trigeminal system - the entrance zone of the trigeminal nerve root to the pons - with the obligatory pathological functioning of the central nervous system. Compression and deformation of the nerve root by vascular formations detected during microsurgical interventions are the cause of trigeminal neuralgia in the vast majority of cases. Elimination of pain after microvascular decompression of the parastem part of the trigeminal nerve root served as the basis for creating a hypothesis about local demyelination of nerve fibers caused by arterial and venous structures. In the work of Yu. A. Grigoryan, light microscopy performed on the surgical material of 7 patients with clinical trigeminal neuralgia syndrome showed that the primary factor damaging the nerve fibers is compression of the root from the outside by various vascular formations and tumors.

Infection with herpes viruses, determined, incl. and in the local population [S.G. Mardanly et al., 2019] by the presence of specific IgG, depends on the type of virus and for HHV-1, HHV-3, HHV-4 and HHV-5 reaches 90% [1]. However, neutralizing antibodies that persist throughout life often do not

prevent possible relapses. An increase in morbidity associated with herpes viruses during the COVID-19 pandemic, identified in a number of studies, may be associated with stress and weakened immunity of victims during the infection. It is known that during infection, COVID-19 has a direct effect on T lymphocytes, leading to immune dysfunction and lymphopenia with a decrease in the body's resistance to infections and reactivation of herpes viruses [2].

According to Bakaev V.V., Mardanly S.S. (2022) in connection with SARS-CoV-2 infection, cases of the development of manifestations of herpes zoster (VZV), neuralgia, rashes and ulcerations of the mucous membranes characteristic of HHV1 have been established [3,4]. It is well known that COVID-19 is also dangerous due to subsequent complications, including depression, irritability, insomnia, memory impairment, fatigue, weakness, impaired taste and smell, collectively known as “post-Covid syndrome” [5]. It has been established that reactivation of herpes viruses can also occur in the post-Covid period, demonstrating skin rashes, damage to mucous membranes and more serious symptoms in patients, including chronic fatigue syndrome (CFS) and depression, caused by herpes viruses types 6 and 7 [6].

The general symptoms of neuropathy of the facial nerve in COVID-19 are paralysis of the entire facial muscles [20]: the frontal folds disappear, the corner of the mouth is lowered, the eyebrow is raised upward, due to paralysis of the orbicularis oculi muscle, there are no full blinking movements and complete closure of the eyelids (lagophthalmos). Bell's symptom is often observed, which consists in the fact that when you try to close your eyes, the cornea is visible and the eyeball on the paralyzed side deviates upward and outward. Involvement of the trigeminal nerve in the pathological process is characterized by hypoesthesia and sometimes neuralgia [17]. With pathology of the trigeminal nerve, the lack of sensitive innervation leads to the fact that, as a result of the irritating effects of environmental factors on the conjunctiva or cornea, protective blinking movements do not occur and the cornea remains naked, resulting in damage to the epithelium. Also important is the fact that there is no neurotrophic component in the development of corneal complications.

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