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Post-Covid Facial Prosopalgia

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Annotation: For many decades, prozoopalgia has baffled specialists due to the versatility of its clinical manifestations and the lack of an orderly classification of the disease. In recent years, the number of studies and publications on the problem of pain in the facial area has increased manifold. This situation is associated not only with difficulties in diagnosis, treatment or polymorphism of facial pain, but also with the lack of clear criteria for an interdisciplinary approach to patient management. This article provides a review of the literature on modern concepts of post-Covid trigemenal neuralgia.

Keywords: prozoopalgia, facial pain, coronavirus, pain syndrome.

Relevance. Today, trigeminal neuralgia (TN) is considered one of the most common prosopalgia and is a typical example of neuropathic pain of a paroxysmal nature. According to various literature sources, trigeminal neuralgia (TN) occurs with a frequency of 4–13 cases per 100,000 population. NTN manifests itself as very painful and excruciating facial pain and is one of the most persistent pain syndromes. This type of facial pain often has a recurrent or chronic course and is very difficult to treat compared to other types of facial pain. Therefore, trigeminal neuralgia has a negative impact on the quality of life of patients, leading to disability.[1] Today, trigeminal prosopalgia is a chronic disease that manifests itself as intense, burning, shooting neuralgia in the area of innervation of one or more branches of the trigeminal nerve.

For many decades, prozoopalgia has baffled specialists due to the versatility of its clinical manifestations and the lack of an orderly classification of the disease. [12]. In recent years, the number of studies and publications on the problem of pain in the facial area has increased manifold. This situation is associated not only with difficulties in diagnosis, treatment or polymorphism of facial pain, but also with the lack of clear criteria for an interdisciplinary approach to patient management [2, 3].

Chronic facial pain due to trigeminal neuralgia (TN), one of the commonly diagnosed causes of facial pain, is characterized by severe lancinating pain paroxysms with a remitting course. With a long course of the disease, specialists are faced with the appearance of interictal facial pain in patients, including a change in the symptom complex of the attack itself, which does not fit into the clinical picture of the disease.

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.

The development of trigeminal neuralgia proceeds as follows: compression of the Gasserian node by tortuous and dilated vessels leads to demyelination (destruction of the myelin sheath) of the nerve fiber; impulses spreading to the trigeminal nerve and nearby nerves cause prosopalgia. As you know, the trigeminal nerve is the fifth pair of cranial nerves; it is formed by three branches: ophthalmic,

maxillary and mandibular. The clinical picture of the disease is determined by. Which branch is affected? Trigeminal neuralgia is a syndrome that includes a wide variety of clinical symptoms. These include: ♦ shooting, burning pain on one side along the affected nerve fiber, which lasts up to 3 minutes; ♦ spasm of the facial muscles throughout the attack; ♦ dilated pupils; ♦ increased separation of tear fluid and saliva; ♦ immobility of the patient during an attack; ♦ the appearance of prosopalgia when touching the skin of the face. Prosopalgia can persist in patients for several days, while in the initial stages the pain is less pronounced, it is short-lived and almost invisible to the patient. The peak of pain occurs during an attack, which occurs spontaneously. In addition, an attack can be triggered by exposure to trigger points, which are most often located in the area of the nasolabial triangle.

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.

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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