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MODERN ASPECTS OF DIAGNOSTICS AND TREATMENT OF FACIAL PAIN

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Abstract:

Atypical facial pain is the least studied variant of prosopalgia (facial pain), since several mechanisms are involved in its development, and the psychogenic component is considered the main one. The disease is most often associated with depression, anxiety disorders, and other mental illnesses. To date, other predisposing and provoking factors of pathology have been established.

Keywords: nociceptive, neuropathic, psychogenic, dysfunctional, odontogenic pain, chronic stress.

INTRODUCTION. The most common causes of nociceptive facial pain are diseases of the teeth and periodontal tissue. Odontogenic pain is one of the most frequent and painful. It is capable not only of irradiation, but also of repercussion (reflection into other zones). Few dental diseases are chronic, but given their high prevalence, they need to be diagnosed in patients with chronic facial pain. So, it is known that with the defeat of the wisdom tooth or even its difficult eruption, pain can be felt in the ear and the TMJ area. When the molars of the upper jaw are affected, pain may occur, spreading to the temporal region, the upper jaw. The lesion of the molars of the lower jaw can cause pain reflected in the larynx and crown, sublingual area. In the pathology of incisors, pain is usually reflected in the nose and chin area. There is also intraoral pain that is not associated with dental tissues (with damage to the mucous membrane of the oral cavity, tongue, periodontal tissue). The literature describes cases of nerve damage either due to dental procedures or due to trauma. Recently, an international group of experts proposed using the term "permanent dental pain disorder" to classify permanent pain without a local disease (there are other names - "atypical odontology" or "phantom toothache"). Terms such as posttraumatic trigeminal neuropathy, peripheral painful traumatic trigeminal neuropathy can be used in cases where there is a clear correlation between trauma and the development of pain.

Episodic neuropathic pain includes paroxysmal neuralgia, of which the most frequent is trigeminal neuralgia (TN). The main reason for its development is the local demyelination of the trigeminal nerve due to mechanical compression (vascular) or an autoimmune process, for example, in multiple sclerosis. In the second case, local demyelination and the formation of efaptic transmission are morphologically detected; it is known that TN may be the only early sign of multiple sclerosis. The epidemiological feature of TN is that its frequency is higher in women and elderly patients. The clinical picture of TN is characterized by short paroxysms of burning, shooting, lightning-fast pain (duration - several seconds), usually provoked by irritation of trigger points in the face area.

However, this is often limited only to the branch of the nerve involved in the pain syndrome. A refractory period (from 30 seconds to 5 minutes) and antagonistic gestures are characteristic when the trigger zones are irritated immediately after the end of the previous attack. The features of this pain syndrome are the contraction of the muscles of the corresponding half of the face and the pulling of the corner of the mouth. These muscle contractions are designated as tonic and clonic convulsions resulting from the transmission of excitation from the trigeminal nucleus to the facial nerve.

Paratrihеminal Raeder syndrome is singled out separately, in which the orbital and maxillary branches of the trigeminal nerve are more often affected in combination with the fibers of the anterior sympathetic plexus. One of the most serious causes of this syndrome is a tumor in the immediate vicinity of the Gasser node. At the same time, the nature of pain differs from classical neuralgia: attacks are very sharp, shooting in the eye socket area on the affected side, more often begin in the morning and last continuously for up to several hours, sometimes accompanied by nausea or vomiting and Bernard —Horner syndrome.

Tolosa—Hunt syndrome (painful ophthalmoplegia). The disease was described by F.J. Tolosa (1954) and, in more detail, W.E. Hunt (1961). Currently, the cause of this syndrome is considered to be an infectious and allergic lesion of the dura mater in the upper orbital fissure with the involvement of the orbital, diverting,

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block, oculomotor nerves, the wall of the internal carotid artery (periarteritis) and the sympathetic fibers lying on it. The picture of the pain syndrome has a bright vegetative color, pain of a permanent nature in the forehead, eye socket, retrobulbar space are described as "cerebral", "gnawing", "drilling". Sensitive disorders are often detected, such as symptoms of prolapse — hyposthesia in the area of innervation of the I branch of the trigeminal nerve. Occasionally, when the process spreads to the top of the eye socket, there is a decrease in visual acuity with signs of atrophy on the fundus. The duration of the disease ranges from several days to weeks.

SUNCT-syndrome (English Short-lasting, Unilateral, Neuralgiform headache with Conjunctival injection and Tearing) is a short—term unilateral, neuralgic-type headache with redness of the conjunctiva of the eye and lacrimation. This rather rare form of primary headache was first described by Norwegian researcher O. Sjaastad in 1978. SUNCT syndrome is characterized by paroxysmal, unilateral pain localized peri- or retroorbitally, lasting about 60 seconds, accompanied by local autonomic disorders. It is known that this disease most often occurs with remissions and exacerbations: up to 20 pain attacks are noted per day, most of which occur during wakefulness, although nocturnal attacks of pain are also possible (1.2%).

Neuralgia of the tympanic nerve. It was described in 1933 by F. Reichert. It is quite rare. It is considered as a kind of neuralgia of the pharyngeal nerve, which is due to its anatomy. The etiology and pathogenesis are not reliably known. The localization of pain is the external auditory canal, the TMJ on the side of the lesion and the area adjacent to it, sometimes in the depth of the ear. Pain paroxysms with this pathology occur spontaneously, without provoking factors, are characterized by the absence of trigger zones; at the same time, after an attack, patients note itching and dull pain in the external auditory canal.

Myogenic pains and myofascial pain syndromes. It is known that myofascial disorders in the facial musculature develop by the same mechanisms as in skeletal muscles, and cause 10-20% of pain syndromes on the face. With prolonged fixed local hypertension, secondary local disorders occur in the masticatory, temporal and pterygoid muscles, such as vascular, metabolic, inflammatory, they are regarded as trigger points. The most common etiological factors that cause myofascial disorders on the face include malocclusion (Kosten's syndrome), characterized by pain in the TMJ and specific sound signals in the inner ear (squelching, squeaking, ringing) that occur when opening the mouth and swallowing, as well as reflected pain from the muscles of the neck and upper shoulder girdle and all a well—known psychophysiological phenomenon is bruxism.

Psychogenic pain. Psychogenic pain is a diagnosis of exclusion. Facial psychalgia can occur with depressive disorder, including "masked" depression. The pathogenesis of this type of pain is not completely clear, probably there is a decrease in the activity of the antinociceptive systems of the brain. In the treatment of such patients, cognitive behavioral therapy is used, as well as various relaxing techniques.

Atypical facial pain is a chronic pain in the face that does not meet the criteria of other cranial neuralgias, is not associated with somatic pathologies. According to modern diagnostic criteria, atypical pain should be observed daily for at least two hours a day for three months or longer. The diagnostic program involves consultation with a psychiatrist and neurologist, dental examination, X-ray examination of the bones of the facial skull. Treatment includes psychotropic medications (antidepressants, anticonvulsants), cognitive behavioral psychotherapy, hardware methods of exposure.

The term "atypical facial pain" has existed since 1924, when it was proposed by doctors S. Fraser and

I. Russell to describe atypical trigeminal neuralgia. Since 2001, the name has been replaced by the modern term "persistent idiopathic facial pain" (PILB), which more accurately describes the nature of the pathology. The prevalence of facial pain in the general population is 17-26%, and in 6% of cases the malaise is caused by PILB. Up to 60-70% of those suffering from atypical pain are middle-aged women.

Atypical facial pain is the least studied variant of prosopalgia (facial pain), since several mechanisms are involved in its development, and the psychogenic component is considered the main one. The disease is most often associated with depression, anxiety disorders, and other mental illnesses. To date, other predisposing and provoking factors of pathology have been established:

> Chronic stress. Psychoemotional tension triggers disturbances in the interaction of mediators, as a result of which the balance between the nociceptive and antinociceptive systems of the central nervous system disappears. Stress factors have a particularly negative effect on people with a passive coping strategy (a passive approach to countering stress).

> Surgical manipulations. Dental treatment, maxillofacial surgery, plastic surgery may be accompanied by damage to peripheral nerve fibers, which triggers the process of phantom soreness.

> Facial injuries. If PILB was observed before the injury, then after the injury it usually worsens, and patients mistakenly consider an accident

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to be the main cause of the disease.

> Tension of the masticatory muscles. In most patients, there is an asymmetry of muscle tension, a diffuse increase in muscle strength, excessive compression of the natural bite.

The mechanism of occurrence of atypical facial pain still remains undisclosed. It is assumed that the main role is played by disorders of neurotransmitter regulation in the central nervous system. At the same time, the sensitization of nociceptive fibers increases, the phenotype of afferent neurons changes, nerve cells are cross-activated, as a result of which signals can be transmitted without the participation of neurotransmitters.

Atypical pain and psychogenic factors are characterized by complex causal relationships. On the one hand, a long-term pain syndrome reduces the quality of life, provokes negative emotions, causes the formation of anxiety, depression. On the other hand, changes in the exchange of neurotransmitters in mental disorders can cause chronic painful sensations in the face.

The main complaint of patients is facial pain, which varies significantly in nature and intensity. Patients describe it as dull, aching, deep, exhausting. Some people describe the sensations as painful or unbearable, but most note the average intensity of the pain syndrome. Atypical pain in the face has a chronic course, increases under the influence of triggers, but does not have the character of attacks, unlike trigeminal neuralgia.

Atypical soreness becomes stronger under the influence of cold, stress, dental manipulations. Sometimes it migrates from one part of the face to another, in 40% of cases it appears bilaterally.

Cases of discomfort spreading to the neck and shoulder girdle are described. Facial pain persists all day, periodically weakens or intensifies. At night, most of the patients do not have it. More than 65% of patients experience a feeling of numbness in the facial area, "crawling goosebumps", hot flashes, and other unpleasant non-painful sensations. Often there is a subjective feeling of puffiness or asymmetry of the face, although an objective examination does not detect this.

A characteristic feature of atypical pain is the absence of sensory disturbances when assessing the neurological status of the patient.

Chronic pain syndromes in most cases have several localizations. In addition to atypical prosopalgia, patients are concerned about soreness in the back and lower back, unexplained myalgia, migraines. Many women face moderate or severe premenstrual syndrome, dysmenorrhea. Somatoform disorders are represented by irritable bowel syndrome, neurodermatitis, hyperventilation syndrome.

Psychiatric diseases are more common among patients with idiopathic facial pain than in the population. 16% of people are diagnosed with affective disorders, 15% — signs of somatoform disorders, 5% — psychosis. Hypochondria develops against the background of atypical prosopalgia, which is why people turn to many specialists, recognize themselves as seriously ill, and require various, often unnecessary treatment from doctors.

Neurological examination. Atypical type of pain is localized in any areas of the face, regardless of the areas of innervation of the trigeminal nerve. Often the pain is felt in the mouth area. In the study of sensitivity, deviations are not detected, although patients complain of various sensory disorders. There are no trigger points on the face.

> Dental examination. To exclude the odontogenic nature of soreness, a standard examination is recommended. If affected teeth are found, their treatment is necessary, after which in some cases prosopalgia disappears.

> Radiography of the bones of the facial skeleton. The study is necessary if damage to the temporomandibular joint is suspected. Additionally, an orthopantomogram, CT of the skull bones is prescribed. The presence of volumetric neoplasms, other organic lesions of the central nervous system is excluded during MRI of the brain.

> Consultation of a psychiatrist. Since atypical facial pain often has a psychogenic cause, a full examination is mandatory.

> In practice, the criteria of the beta version of the International Classification of Disorders Accompanied by Headache (ICGB-3) are applied. Treatment of atypical facial pain

Clear principles of therapy for atypical facial pain have not yet been developed. The generally accepted tactic is to minimize unjustified dental and surgical manipulations, even if the patient insists on carrying out such. This is necessary to exclude the typical traumatic triggers of pain syndrome.

The medical treatment of the disease is empirical. The following groups of drugs are used:

Y Antidepressants. Tricyclic antidepressants have the best effect, which normalize the level of neurotransmitters in the brain, stimulate the antinociceptive system of the central nervous system.

Y Anticonvulsants. Benzodiazepines and GABA analogues are shown, which have an additional analgesic effect. A combination of anticonvulsant medications with antidepressants shows good effectiveness.

Y Botulinum toxin. With severe facial soreness, it is possible to achieve a temporary analgesic

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effect by injecting botulinum toxin type A into the gums, palate, upper lip.

Y Antibodies to the S100 protein. The innovative drug normalizes the processes of transmission of impulses between neurons, reduces the level of anxiety, increases the resistance of the nervous system to stress.

To enhance the effect of the drugs, non-drug treatment is prescribed: low-energy diode laser, transcranial magnetic stimulation, biofeedback, pulse radiofrequency therapy. Cognitive-behavioral, rational psychotherapy is recommended to reduce anxiety, assist the patient in overcoming pain. In resistant cases, hypnotherapy can be used.

CONSULTATION. Atypical facial pain is a difficult-to-treat disease due to the lack of clinical recommendations, ambiguity of etiopathogenetic features. The treatment reduces the intensity of pain, however, as a rule, atypical discomfort persists or disappears for a short time and reappears under the influence of provoking factors. Taking into account the psychogenic prerequisites of pathology, the basis of prevention is mental health care.

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