TENSION HEADACHE: PSYCHOVEGETATIVE ALTERATIONS AND POSTURE DISORDERS

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Annotation: During the year, 50% of adult Europeans suffering from acholia, as well as at least 90%, complain of headaches once in a lifetime without treatment. This disease has been identified by WHO as one of the 10 diseases leading to disability. With headaches, a high rate of disability is associated with several pathologies that are associated with suffocation in patients. We can bring to them ruxy disorders, neck pain, vestibular symptoms and imbalance, as well as cerebral pathologies in migraines. Having complete knowledge about the disease, knowledge of the mechanisms of formation of the pathological process provides the optimal treatment option. The pathogenetic mechanism of tension headaches (THA), autonomic disorders and posture changes is still being studied and is causing discussion [6].

Keywords: Headache, Treatment, Causes, Symptoms, Condition.

Introduction

THA is a common disease among adults, it affects from 30% to 78% of the population, and this indicator varies around the world [1]. The prevalence among the European population is 60% [5], the indicator is higher among women (in the ratio of 4:5) [5]. The prevalence depends not only on age, but also on race and level of education [6].

THA is one of the most urgent problems of modern medicine. According to World Health Data, THA is the most common among neurological diseases, ranking 3rd among the causes of disability .In the scientific literature, 85% of the population with this pathology experience periodic headaches, 40% of them experience a decrease in social and labor activity. activity, and it is mentioned as requiring qualified medical care [3]According to Russian cephalologists, the occurrence of THA is 78% [1.]

It was found that in middle-aged people, episodic tension headaches (ETHA) occur in 18%. Various studies have found that tension headaches (THA) are observed in a third of the population aged 30 to 39 years. According to the World Health Organization, three quarters of the population aged 18 to 65 years have had at least one headache attack in the last year, while chronic tension headaches are observed in 2-3% of the population. It was found that THA occurs 3 times more often in women than in men [18]. Despite the prevalence of headaches in the population, the diagnosis of THA is made only in 1% of cases at the first consultation with a doctor [8].

While episodic forms of THA are not considered a serious medical or social problem, THA is accompanied by various concomitant disorders - depression, sleep disorders, somatoform disorders - that distort the patient's daily life and quality of life. The difficulty of choosing an effective treatment brings NWBO to the level of a complex socio-medical problem [2].

As a result of the development of modern diagnostic methods, views on the neuromorphology and neurophysiology of THA have evolved [1]. The forms of THA vary to this day[2]. Despite numerous studies conducted in this regard, there are still many aspects that can cause mucocams in relation to the pathogenesis of the disease [8]. Today, vascular, neurogenic, psychogenic, biochemical and myofascial theories are considered as the main pathogenetic mechanisms of THA. The term THA was coined in 1963 H.It It was introduced into science by Wolf, who made a patient's observation about the ongoing tension in the head and skeletal muscles in as a result of the devastation caused by the disease [13]. As a result of persistent spasm, there is a feeling of pressure on the head, squeezing, wearing a "helmet". The tense muscle caused vasospasm, leading to ischemia, as well as an increase in unpleasant hissing. Circulatory disorders manifest themselves in the form of darkening of the veins, which leads to the emergence of a vicious circle leads to the formation of. As a result, the muscle cannot be well supplied with blood, as a result of its tension, residual metabolic products accumulate, the muscle swells again, unable to exit through

the venous outflow, the pain increases. EMG tests show that people with bo have higher electromyographic activity than healthy people. Muscle tension bo is strongly expressed in the existing Sox. This is based on the findings of EMG that muscle tension is of paramount importance for the formation of STHA. Muscle tension and cervicogenic factors occupy an important place in the formation of NWBO. The presence of connections between the area of distribution of BO and a specific muscle corresponds to myofascial pain [5]. Myofascial pain is characteristic of THA, migraine, cervicogenic bo. Trigeminovascular and trigeminoservical mechanisms are also involved in the formation of bo [2, 6]. Ferguson L.U. and co-authors claim that the presence of a large trigger system of nerves ensa can lead to the generalization of pain in the frontal chakra [5]. Collaterals of 3 branched nerve afferent fibers fall on the three upper cervical roots. Thus, when exposed to S1-S3, the 3-branched nerve is also affected, which is manifested by pain, thus forming one of the peripheral mechanisms of the formation of STHA [3]. As a result of activation of nociceptors of the spinal column, muscular and pineal glands, roots and nerve columns, signs of pain in the cervical cranial region are observed, neuralgia of the ensa nerve and pericranial THA muscles are manifested along with signs of damage [1, 7]. Taking into account the importance of musculotonic syndrome in the pathogenesis of the transition of THA to a chronic form, it is necessary to take into account measures for the treatment of overstressed pericranial muscles [3]. In a number of scientific papers, it has not been proven that there is an inextricable link between an increase in the tone of the frontal muscles and the intensity of bo, therefore, the authors believed that muscle tension has no significance in the pathogenesis of po [2]. A study by Marcus, MD, showed that there were no significant differences between electromyograms of the THA face and neck muscles and healthy people [9]. T.A. A study by Ahles and co-authors showed that there were no significant differences between muscle tension in different types of bo [5].

Studies by V. Pfaffenrath and W. Gerber showed that in some patients with THA

there is no tension in the extracranial muscles, while in another group it is caused by a reaction to stress or direct bo [10]. Studies by A. Hopkins and D. Ziegler have shown that the muscle of the scalp cannot serve as a basis for the diagnosis of THA [8]. Based on this, it was shown that the electromyographic activity in migraine was higher compared to THA. Contradictions in thoughts formed the basis for the division of patients with THA into 2 groups: types that are accompanied by tension of the pericranial muscles, and without tension of the pericranial muscles [2]. Tingling in the THA pericranial muscles served as a source of nociceptive afferentation, as well as pain in the vast majority of patients present [7, 3]. M.V. Ryabus (1998) [4], O.A. Kolosova (1999) [9], E.Ya. Strochunskaya (1996) [2] studied the properties of electromyographic activity in her work. During EMG testing, an increase in the muscular potential of the oral cavity was observed in patients with NWBO. R. Studies by Sherman and co-authors showed that pain is not always observed at a time when the muscle is tense, and that pain does not always decrease after the tension has passed. It has been shown that the relationship between muscle tension and pain is clear and persistent. Even mild muscle tension can lead to the release of biologically active substances, which leads to increased headaches. The importance of nearby large muscles - the trapezius muscle - should be taken into account [8]. The authors believe that prolonged muscle tension leads to an increase in potassium levels, damage to chemoreceptors in the Nati, causing headaches. Vasoconstrictors when using Bo, there is an ischemic cause in the suppression of the carotid arteries. Patients experience narrowing of the intracranial arteries[3].

The authors believe that during the formation of THA there is a violation of the vascular component[5, 6]. Violation of management arterial and venous cerebral hemodynamics is complicated by compensatory muscle spasm, especially observed in life-threatening stressful situations, which leads to a deterioration of venous outflow. A study by Yakubenko Yu.V. (2015) showed a paradoxical vascular reaction - a decrease in the linear velocity of blood flow in patients with the presence of NWBO. This case indicates a violation of the autoregulation of

cranial vessels caused by chronic persistent distress and compensatory muscle spasm [5]. A modern morphological study of the underlying subcortical systems has shown the presence of effects of serotonin and noradrenaline systems on the trigeminal vascular system[8]. Axons of neurons, along with cells of the trigeminal vascular complex, are connected to the vessels of the ventricle of the brain, which not only deliver sensory information, but also affect the intensity of cerebral circulation[14, 18]. As a result of the increased activity of neurons, the meningeal vessels sharply narrow, the sensitive ends of the tricorn nerve are affected, compensatory vasodilators are released to normalize vascular tone, aseptic inflammation is observed [11]. Studies by

V. Pfaffenrath and W. Gerber have shown that changes in the reactivity of the limbic-reticular system and dysfunction of endogenous mechanisms of antinoceptive control lie in the pathogenesis of THA [15]. The antinoceptive system is located in various parts of the central nervous system (gray matter (PAG) on the periphery of the tap, the nucleus of the brainstem, the nucleus of the reticular formation, thenucleus of the thalamus, the inner capsule, the cerebellum, the interneurons of the spinal cord, and so on.qlar), demonstrating the effect of inhibition of nociceptive afferent waves emanating from the dorsal horn of the spinal cord. In Bo, serotonin, noradrenaline, GABA, and opium systems are involved [19]. The participation of nociceptive and antinoceptive systems in the formation of THA is confirmed by psychovegetative disorders, EMG data [9]. The influence of peripheral nociceptors is directly related to the functional state of the higher nervous system. Depression and anxiety in THA occur in high percentages of cases [19]. Wayne A.M. his research has shown that chronic emotional stress is of great importance in the formation of THA. Emotional stress leads to THA when psychological chemotherapeutic mechanisms do not work well in people with certain personality traits, while the antinociceptive system does not work well [17]. The author believes that the inclusion of the limbic system and the higher nociceptive system in the process leads to autonomic-endocrine and psychomotor activation, manifested by

increased muscle tone, ischemia, muscle edema and biochemical changes [7,8,]. The views of A.M. Wayne were reflected in the works of other researchers in the field of the future [11]. E.Ya. Strachunskaya came to the conclusion that the neurotic nature of the personality in the formation of THA and the presence of chronic stress leads to a violation of the functional state of the limbic-reticular system [2]. Bo is caused by hypertension of the pericranial and mimic muscles and hypoxia caused by changes in the functioning of the nociceptive and antinoceptive systems, anxiety- depressive syndrome and changes in the thyroid gland [3, 4]. N. Marlowe and Hammuallifi studied the central mechanisms of bo by evaluating potentials called somatosensory [9]. The results of the study showed that patients with THA had a higher P-1-n-1 amplitude than healthy people. In the N-1-P-2 study, no specific changes were found between seizures and seizures in patients with Bo. In addition, no differences in Ham were found in patients with migraine and THA. Studies by Kuznetsova E.A. and Yakupova E.Z. (2012) have shown that the results of visual, acoustic, somatosensory, cognitive, so- called trigeminal nerve potentials change the activity of the segmental surface system in STHA, as well as age-related changes [11, 12]. Lapina S.E. and Belyakov K.M. Investigated skin sympathetic potentials induced in existing patients. Also, in the Nakatani method, the autonomic nervous system was evaluated for specific characteristics in its management. The results of the study showed that the parasympathetic nervous system prevails in patients with THA, the activity of the sympathetic nervous system decreases. Autonomic dysregulation depends on changes in the upper part of the segment [3, 4].

3 Neurons of the sensory nucleus of the horny nerve converge to the endogenous antinoceptive system columnar systems[4, 14]. They, in turn, reduce the flow of pain in the vessels of the trigeminal nerve [9]. At this stage, tap acquired the gray matter of the ventrolateral part of the vIPAG and the ventrolateral region of the elongated brain, the alpha part of the reticular large cell nucleus was acquired by muximu achamiyat. The pathogenesis of BOS is dominated by vIPAG muxime, which constantly monitors the nociceptive system at the segmental level,

controlling all the activity of the antinoceptive system [9]. vIPAG enhances the passage of pain at the spinal level in the 3-branched nervous system, as well as in the ventromedial joint, bilateral anastomoses become characteristic and control the passage of pain [14]. Efferent PAG neurons can be gabaergic and glutamatergic [10]. They are activated, on the one hand, through their GABA-interoneurons [6], and on the other hand, through the glutamatergic system through the ascending spinomesencephalic pathway and the trigemino- and spinothalamic pathways [4]. Pag also identified enkephalinergic autoregulatory interoneurons and endorphin-retaining cells [14, 10]. Consequently, in turn, inhibitory and activating effects are manifested. An increase in the pain flow entering the PAG is manifested in an increase in segmental antinoceptive control [4, 10]. Electrical stimulation of PAG has been found to cause migraine pain attacks[4, 8]. Bo was observed in the form of psychoemotional and vegetative disorders [14]. In patients with Bo and facial pain, this treatment led to an improvement in axvol [9]. PAG has shown that it can not only function as an antinociceptive system, but its activation can also manifest itself in the form of inhibition. A decrease in pain transmission during inhibition is observed segmentally, and this will depend on the final functional state of the trigeminal vascular system [8].

The central mechanisms of chronic THA conversion have been confirmed by researchers, which is Buchgreitz L. And it found its confirmation in the work of hammuallifs [3]. In the studies of the authors, it was found that patients with chronic migraine and NWBO have increased blood pressure, decreased needlework ability, nociceptive flexor reflex, "clockwork" phenomenon [3]. All this shows that the central mechanisms of chronic transformation in various types of bo are significant [16]. A number of authors show that there is a correlation between the amount of serotonin in the blood and the duration of pain syndrome [4]. The episodic form of THA and the content of serotonin in the blood during migraine did not differ from those in healthy people, while in chronic forms of cephalgia, its amount was found to be significantly reduced.

Serotonin plays a role in antinoception, controls affective reactions, controls sleep and wakefulness, participates in vegetative activity, it is these conditions that give clinical signs of disorders of serotonin metabolism [9]. One of the suprasegmental systems involved in the pathogenesis of Bo is the hypothalamus, which occupies a central place in vegetative, endocrine, behavioral reactions [4,9]. The hypothalamus participates in the central process of nociception, ensures the integration of incoming somatovisseral pain flow, as well as efferent vegetative effects. This is his thalamus, PAG, reticular formation, limbic system arises due to its numerous bilateral connections with autonomic preganglionic neurons[4]. Activation of paraventricular nuclear neurons is accompanied by increased secretion of the corticotropic rylation factor, an increase in the level of adrenal stress hormones, and a compensatory chemoadaptation reaction of the body to pain is observed[6]. The authors' studies have shown changes in the daily content of prolactin, melatonin, growth factor, testosterone, orexin A and cortisol in plasma, cerebrospinal fluid and saliva in patients with THA and migraine. This condition indicates the presence of changes in the activity of the hypothalamic-pituitary- adrenal gland [90]. Studies have shown that in women of reproductive age there are reliable pathological connections between the functional state of the pituitary-ovarian system and THA.

Derevyanko H.P. and Speransky V.V. Studies conducted by STHA da Haiz have shown that there is a pronounced imbalance between sex hormones and cortisol, depending on the stages of the cycle. This hypothesis confirms the vascular and neuroendocrine theory of THA formation [17]. There are also theories concerning immune and pain sensitivity in the development of THA, with A.P. Rachina and

A.A. Logvinova, also G.N. Krijanovskaya and co-authors in their research covered all stages of pain syndrome from peripheral hyperalgesia to the manifestation of emotional and behavioral symptoms [3, 4]. Karpova M.I. BO studies have shown that existing patients have changes in the status of cytokine, and also confirmed the absence of additional secondary immunodeficiency. THA

DA showed a positive correlation between IL-1α and TNFa with days of pain. Elevated levels of IL-1a and TNFa are considered a laboratory sign of immunodeficiency. At the same time, the level of TNFa increases even in the absence of clinical signs of pathology of the immune system. With episodic THA, the number of cytokines was not higher than that of healthy people [6]. The cause of cytokine imbalance may also be a genetic predisposition [10], drug abuse 6], stress and depression [2,8]. The role of the immune system in the development of Bo, special immune reactions, as well as autonomy for further research are revealed. The most recent evidence suggests that THA has peripheral and central mechanisms. To date, the pathophysiological mechanisms of THA have been studied.

This multicomponent system allows you to study, diagnose and treat a disease based on the data obtained. The cause of the disease is mainly indicated by the muscles of the head and neck, stress and central sensitization [6]. Peripheral myofascial nociceptors with the development of muscle pain and an acute episode of THA, repeated episodes of muscle pain increase the sensitivity of the central nervous system, leading to THA overstrain. Muscle tension is observed in both episodic and chronic course of the disease [6]. THA can be accompanied by both mild pain and symptoms of disability. Exacerbation of symptoms can lead to loss of working days, decrease in social activity of the patient, depressive state of the patient [7]. Stress and air can also play an important role in the formation of THA [8]. Ambassador et al. Their study showed that THA manifestations have increased in the Middle East and North Africa as a result of increased zinc problems. It is in these states that the level of anxiety, stress and depression is 30% higher than the global average. According to the data provided by the authors, the average THA of these gods is 20.5% [9]. In studies by other authors, when gender differences were studied in patients with THA trigger points, it was found that trigger defects are especially active in women, and it was shown that these points are higher in the areas of Chuck and ensa. It was also found that the sensitivitybias at these points is higher than in men.

It is believed that the episodic type of THA can develop into a chronic form as a result of exposure to several triggers at the same time. It was found that muscle stress, lack of relaxation, stomach ailments, sleep disorders, excessive consumption of drugs in excess of Hadda, lead to an increase in the amount of bo, even to the chronic development of THA [11].

To date, the most common model for explaining the chronic course of the disease is based on sensitization [10]. According to this model, peripheral nociception S1-S3 originates from an active myofascial trigger point (upper trapezius muscle, ensa muscle) and 3 branched nerves (chakka muscle, masticatory muscle) in the upper segments of the neck. If this concept were true, then attacks of non- dull pain passing through the nucleus of the 3rd corneal nerve of the neck would lead to sensitization of the central nervous system. In addition to active myofascial trigger points - suboccipital, upper trapezoidal, chakra, obtuse - vertebral - sucker and pressure-induced hypersensitivity in the muscles ofthe eye area leads to observation [10].

Conclusion:

1. Kinesiopathological model is a state of behavior or habitual position. This model is associated with a change in the nature of contraction and biomechanical changes in the muscles of the neck and skull (forehead, chin, zygomatic, pterygoid, obtuse-sucker, trapezoidal). To date, the mechanism of THA occurrence is difficult to explain using this model, and it would be a mistake to assess the disease by muscle tension [1, 9, 10].
2. Anatomopathological model - combines the mechanisms of the central and peripheral nervous system. Since the peripheral level refers to the muscular myofascial trigger points involved in the process, headaches are caused precisely by stimulation of trigger points. In the neck, cranial and extracranial muscles, sensitivity to pain increases, nausea from pain and nausea tolerance decreases [1 , 6, 11] .
3. Psychosocial model - stress is understood as mental stress. Excessive stress, zinc overload, nausea, negative thoughts and depression lead to THA. It was also

found that the relationship between sleep disorders and THA when studied leads to the fact that THA becomes chronic [11 , 12]. Other factors in the development of THA can be called dehydration, the use of caffeine, tobacco and alcohol, hormonal disorders, bruxism [30]. THA reduces the quality of life of the patient due to limited functions, reducedproductivity. It was found that during the THA attack, the quality of students' products decreased by 24.4% [14].

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