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CHRONIC MIGRAINE: CLINICAL PICTURE, PATHOGENESIS, TREATMENT

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ABSTRACT. Migraine is one of the most well-known and widespread neurological diseases, occurring in the adult population with an average frequency of 12% [Lipton, 2001]. Chronic migraine (CM), being a well-known clinical problem, was identified as a separate form of headache only in 2004 in the International Classification of Headache Disorders, Second Revision. Chronic migraine is classified as a complication of migraine. Migraine, while not a fatal disease, significantly reduces the quality of life of patients [Osipova, 2003]. The World Health Organization has included migraine in the group of the most maladaptive chronic diseases. The financial and economic costs associated with temporary disability, as well as with the diagnosis and treatment of migraine, are enormous and comparable to the costs of cardiovascular diseases [Stovner et al., 2007; Burton et al., 2009].

With CM, headaches become almost daily (more than 15 days per month), which leads to even more severe maladaptation of patients, an increase in direct and indirect costs associated with migraine, which is of great pharmacoeconomic and medical-social significance [Goldberg, 2005; Steiner et al, 2007].

In the general population, patients with CM account for from 0.4% to 2.4% [Castillo, 1999; Katsarava et al., 2008; Scher et al., 2009]. CM is also included in the group of chronic daily headaches - the most difficult form of cephalgia in terms of diagnostics and therapy, where CM accounts for 55% to 87% of all cases [Katsarava et al, 2004; Bigal et al., 2008].

The high prevalence of CM among young people of working age, severe maladaptation and a decrease in the quality of life of patients, socio-economic consequences determine the relevance of the problem [Osipova, Tabeeva, 2007; Latysheva, Filatova, 2008; Bigal, Lipton, 2008]. Particularly It should be noted that there are difficulties in diagnosing and treating this long-known, but recently identified as a separate form of headache [Alekseev, 2006; Rothrock, 2008; Silberstein et al. 2009]. The pathogenetic mechanisms of CM are not fully understood, among which the dysfunction of the antinociceptive systems of the central nervous system, changes in the excitability of the cerebral cortex, peripheral and central sensitization are actively discussed [Latysheva, 2009; Pietrobon, Striessnig, 2003; Goadsby, Hargreaves, 2008; Aurora, 2009]. When determining the risk of developing CM, the following are considered and taken into account: the role of depression, anxiety, abuse of drugs to relieve headache attacks, dysfunction of the pericranial muscles and other factors [Bussone G., 2003; Bigal, Lipton, 2006; Katsarava et al,, 2007; Dodick et al., 2009].

The issues of therapy of CM have not been sufficiently developed: standard preventive treatment regimens for migraine have proven to be ineffective, which required differentiated and pathogenetically substantiated approaches to therapy.

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CONCLUSIONS

For the first time, a detailed clinical and neurological analysis of a representative group of patients with CM was conducted, identifying the clinical features and variants of the disease course. The clinical criteria for CM diagnostics and the list of comorbid diseases and disorders were clarified. For the first time, an analysis of the phenomenon of "background" headache in CM was conducted. The use of a clinical and neurophysiological approach showed that the phenomenon of "background" headache is one of the clinical manifestations of central sensitization.

The study showed that CM is the result of multilevel disorders of the functional state of the nervous system, which can only be assessed with a comprehensive neurophysiological examination. Cortical disorders are manifested by changes in the excitability of cortical neurons of the visual and, to a lesser extent, motor cortex; dysfunction of nociceptive systems at the brainstem level is revealed by changes in the threshold of the RIII component of MP, while a violation of the habituation of the RIII component indicates insufficiency of antinociceptive influences.

Involvement of the peripheral nervous system is confirmed by obligatory long-term

Musculotonic and myofascial disorders in the pericranial and cervical muscles, which indicates sensitization of peripheral nociceptors at this level. For the first time, a significant role of sensitization of peripheral nociceptors of myofascial pericranial structures in maintaining pain syndrome in CM has been shown. Regression of peripheral and central sensitization manifestations with a decrease in pain afferentation from myofascial pericranial structures in response to the introduction of BTA proves the pathogenetic significance of peripheral sensitization in maintaining central sensitization. The effectiveness of preventive treatment of CM does not depend on the presence/absence of the factor of drug abuse for stopping a headache attack. This is true for all evaluated types of therapy aimed at three links in the pathogenesis of CM: the latest-generation anticonvulsant topiramate, acting primarily at the cortical level; the antidepressant of the group of selective serotonin and norepinephrine reuptake inhibitors duloxetine, affecting cerebral antinociceptive systems, and BTA.

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