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COGNITIVE DYSFUNCTION IN CHRONIC MIGRAINE.

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Abstract: Cognitive impairment in chronic migraine is an urgent problem. Patients with chronic migraine have a high prevalence of subjective and objective cognitive impairment, primarily memory and attention loss. Patients with migraine (especially chronic migraine — HMM) often complain of memory disorders, concentration, planning difficulties and a decrease in the speed of information processing. Based on this, we decided to assess the prevalence of objective cognitive deficits in patients with ChM.

Keywords: Migraine, headache, cognitive dysfunction, depression, anxiety.

Introduction. Chronic migraine is a condition in which headache occurs more often than 15 days a month for three consecutive months or more. This is an exacerbation of episodic migraine, which is characterized by the appearance of constant headaches for several weeks a month.Migraine symptoms include intense, paroxysmal headaches, often on one side of the head, throbbing and intensifying with physical exertion. During an attack of pain, there may be nausea or vomiting, as well as hypersensitivity to light or sounds.Chronic migraine can significantly worsen the quality of life, leading to social maladaptation and disability.

Methods. The study involved 60 patients with ChM and 20 patients with low-frequency episodic migraine (EM) (maximum 4 days with headache per month). The study was carried out in the clinic of ASMI in the Department of Neurology. All patients were recruited according to the criteria: a) age 18-59 years; b) history of ChM or EM according to the International Classification of Headache Disorders - III beta. The diagnosis was made by a neurologist-headache specialist during the patient's consultation; c) written informed consent. Depression and anxiety were assessed using the Hospital Anxiety and Depression Scale (HADS). HADS defines depression/anxiety as absent at 0-7 points, subclinical at 8-10 points and clinical at more than 11 points.

Cognitive function was assessed using the Montreal Cognitive Assessment (MoCA), the Digit Character Replacement Test (DSST), the Ray Auditory Verbal Learning Test (RAVLT) and the Perceived Deficits Questionnaire (PDQ-20).

The exclusion criteria for all groups were serious mental disorders (with the exception of mild or moderate depression and anxiety), taking benzodiazepines, antidepressants and anticonvulsants (these drugs had to be discontinued at least two weeks before the study) or taking "life-saving" drugs. medications within six hours before the start of the examination.

Results. The study included 60 patients diagnosed with ChM (48 women and 12 men). 20 patients with low-frequency EM (16 women and 4 men) were also included. A total of 67.4% of patients had concomitant headache caused by drug abuse (HGB). More than 90% of these patients used triptans, while the rest abused codeine-containing analgesics or triptans in combination with combined analgesics.

Patients with ChM had higher levels of depression and anxiety compared to patients with EM. However, since subjects with clinically significant depression/anxiety were excluded from the study, both of our groups demonstrated the absence of depression as defined by HADS. Anxiety has reached a subclinical level in the HMM population

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Compared with the participants of MI, the patients of ChM demonstrated higher subjective cognitive impairment, measured on the PDQ-20 scale. Interestingly, the PDQ-20 scores did not correlate with any of the objective cognitive tests (DSST, RAVLT, or MoCA), but positively correlated with the level of depression and anxiety (Spearman's rho = 0.38 and 0.24, respectively).

The ChM subjects had significantly lower DSST scores. At the same time, 28.5% of patients with ChM and only 13.6% of patients in the control group had DST in the lower quarterly range (p=0.04).

In patients with ChM, the overall RAVLT learning score was also significantly lower compared to the control group with low-frequency EM. In addition, the total learning score did not reach the previously published thresholds for the corresponding age group in both groups (p=0.0001 for both groups). Patients with ChM had 4 times higher chances of achieving a total RAVLT learning score in the lower quartile range compared to the EM cohort (odds ratio [OR] 3.8; 95% confidence interval [95% CI] 1.5–9.6; p = 0.005).).

MoCA results were lower in patients with ChM compared to patients with EM, but within the normal range in both groups. Nevertheless, 18% of patients with ChM and 6.8% of the control group scored below the threshold of mild cognitive impairment (26 points) even with no or almost no pain (p=0.09). Patients with ChM had the most severe memory/delayed recall disorders (65.3%), attention (46.5%), abstraction (30.6%) and speech (27.1%).

We did not observe any clinically significant correlations between neuropsychiatric parameters and the results of objective cognitive tests. Interestingly, anxiety correlates positively with RAVLT and MoCA indicators, which suggests that mild anxiety may even be useful for improving cognitive functions.

Correlation between clinical parameters, cognitive indicators and behavioral indicators in patients with chronic migraine.

In order to further study the effect of neuropsychiatric and other parameters on cognitive status in patients with ChM, DSST indicators (as a common indicator of several cognitive functions) were analyzed using multiple linear regression. It was found that years of study and chronic headache affect the DSST score (p=0.02 and p=0.04, respectively). Depression and anxiety had no effect on cognitive functions.

Discussion. This study was aimed at studying the cognitive profile in ChM. Over the past decade, there have been data on pronounced cognitive deficits in migraine patients without aura, both ictally and interictally. During a migraine attack, cognitive impairment was observed at every stage, including the painless phases of the pro- and post-dromal period. Moreover, it has been shown that the severity of interstitial cognitive deficits correlates with the frequency of headaches. This study shows that patients with ChM are characterized by significant cognitive impairment even during the mildest headache or in the absence of headache.

The results of PDQ-20 show that a significant number of patients with ChM have a subjective decrease in cognitive functions. However, these reports correlate with depression rather than objective tests, meaning that patients who do not complain about cognitive problems at work and at home may actually have serious impairments.

It has been proven that patients with ChM have significant impairments in various aspects of cognition, including "complex attention", which was measured using DSST, memory using RAVLT

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and other areas, including language and abstraction, as evidenced by MoCA. These differences were found in comparison with subjects with low-frequency EM. It is assumed that cognitive changes during a migraine attack are caused by reversible brain dysfunction. Our results seem to confirm this hypothesis. This may explain why patients with ChM experience permanent cognitive impairment, even if they have no pain.

It is noteworthy, however, that patients with low-frequency EM also have a certain level of cognitive impairment. For example, in RAVLT, these patients scored below the accepted threshold for subjects aged 30-39 years (55.9 words). This means that brain dysfunction during migraine attacks may not be completely reversible and may become very stable during headache chronization.

Similar cognitive changes are described in major depression and are even listed as diagnostic criteria for major depression in the DSM-5. Since depression is often combined with migraines and other types of chronic pain, it is natural to assume that cognitive impairment in this patient population is at least partially caused by depression.

In this study, we included patients with ChM with mild, moderate depression or no depression to study the etiological role of other factors. No correlation was found between the levels of depression and anxiety and any objective indicators of cognitive activity. Moreover, the presence of ChM in the study, and not depression, was an independent risk factor for worsening DSST results. The present study shows that neuropsychiatric parameters cannot be the exclusive cause of cognitive dysfunction in chronic pain.

The chronification of migraine is closely related to central sensitization (CS), which develops during each attack and gradually becomes continuous with the chronization of pain. Maladaptive neuroplasticity has been described in the brains of patients with chronic pain and ChM, including near-conductive gray, pale ball and striatum, strengthening their connection with other areas responsible for nociception and cognition (prefrontal cortex, anterior cingulate gyrus, amygdala and insular cortex). increased excitability and eventually causes atrophy of gray matter. This may explain why cognitive deficits also become chronic and can be detected after pain resolution and the end of the postdromal period. Moreover, it is not yet known when this maladaptive process will become irreversible, as a result of which some patients with ChM will have persistent cognitive impairments and they will become immune to headache treatment. Previously, it was shown that similar cognitive changes in depression persist in about half of patients even during remission.

In the light of these results, the timely initiation of preventive treatment of EM is justified to prevent ChM, chronic headache and persistent cognitive impairment.

Conclusion. Thus, patients with ChM have significant performance impairments in several cognitive areas, including memory and attention, during the mildest pain and in the absence of pain. Cognitive deficits are stable and are not associated with migraine exacerbations. Chronic pain (and level of education), rather than clinical parameters or depression, are independent predictors of cognitive decline in patients with ChM. Cognitive impairment in ChM can be caused by maladaptive neuroplasticity in the brain, areas responsible for nociception, antinoception and cognition These data confirm the importance of timely preventive treatment of EM. DSST and MoCA are easy—to-use and widely available tools for time-consuming cognitive testing in patients with migraine.

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