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CLINICAL, NEUROLOGICAL, AND CEREBRAL HEMODYNAMIC FEATURES IN PATIENTS WITH CHRONIC MIGRAINE

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Abstract

Chronic migraine (CM) is a complex neurovascular disorder characterized by recurrent headache episodes accompanied by neurological dysfunction. The aim of this study was to evaluate the clinical symptom dynamics, neurological indicators, and cerebral hemodynamic parameters in patients with chronic migraine. The examined patients were assessed for headache duration, intensity, pulsation characteristics, associated symptoms (nausea, photophobia, phonophobia), and trigger factors. Neurological evaluation included cognitive function assessment, signs of autonomic dysregulation, somatovegetative changes, and condition-dependent focal symptoms. Cerebral hemodynamic analysis was performed using Doppler ultrasonography and functional neuroimaging methods to assess brain stability, blood flow velocity, and cerebrovascular reactivity. The results confirmed that interactions between central and peripheral neurovascular mechanisms play a key role in chronic migraine and provide a basis for optimizing both diagnostic and therapeutic approaches.

Keywords

chronic migraine, headache, neurovascular mechanisms, neurological symptoms, cerebral hemodynamics, Doppler ultrasonography, cognitive function, cerebrovascular reactivity, physiological triggers.

Introduction

Chronic migraine (CM) is one of the most prevalent and disabling headache disorders in neurology. According to the World Health Organization, migraine affects 12–15% of the general population, with 2–3% progressing to the chronic form. Chronic migraine is typically defined by headache episodes lasting more than 15 days per month, of which at least 8 days exhibit classic migraine features. Unlike episodic migraine, chronic migraine is associated with long-term neurovascular dysfunction, central sensitization, and alterations in cerebral blood flow.

Clinical manifestations in CM extend beyond headache and may include photophobia, phonophobia, nausea, autonomic responses, impaired concentration, and sleep disturbances. Prolonged pain stimulation leads to functional changes in the trigeminovascular system, indicating the involvement of both neuronal and vascular factors in disease pathogenesis. Additionally, cerebral hemodynamic disturbances—particularly decreased blood flow velocity and reduced cerebrovascular reactivity—may correlate with chronic pain severity and clinical symptom intensity.

A comprehensive analysis of neurological and hemodynamic parameters allows for a better understanding of CM pathogenesis, improves diagnostic algorithms, and has practical significance in selecting individualized therapeutic strategies. Therefore, evaluating clinical signs, neurological status, and cerebral hemodynamic function in CM patients is a relevant scientific and practical task in modern neurology.

In this study, a total of 106 patients with chronic migraine were analyzed from a clinical and neurological perspective, including 86 women (81.1%) and 20 men (18.9%). The mean age of patients was 38.7 ± 0.94 years, with women averaging 38.5 ± 0.79 years and men 39.2 ± 2.69

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years, indicating that chronic migraine predominantly affects individuals during their active working age (30–50 years), with a higher prevalence among women.

Regarding the type of migraine attacks, 35 patients (33%) had migraine with aura, while 71 patients (67%) had migraine without aura, indicating the predominance of the latter in chronic cases. Among women, the proportion of migraine with aura was 0.35 ± 0.05, and without aura 0.65 ± 0.05. In men, migraine with aura accounted for 0.25 ± 0.1, and without aura 0.75 ± 0.1, demonstrating that migraine without aura is more common across both sexes.

The frequency of attacks and clinical manifestations are important indicators in assessing chronic migraine. Patients were categorized into three groups based on monthly headache recurrence: less than 15 attacks per month, 15 or more attacks per month, and at least 8 migraine days per month. These criteria enabled evaluation of disease chronicity and clinical severity.

Chronic migraine affects not only the headache itself but also induces complex neurobiological changes in both the central and peripheral nervous systems. Its underlying pathological mechanisms involve not only pain pathways but also neuronal adaptation, blood flow alterations, and neurotransmitter imbalance. Patients exhibit persistent sensory, motor, cognitive, and emotional changes, which lead to reduced quality of life and shorter interictal remission periods.

Analysis of Data Indicates That the Most Common Neurological Disturbances in Patients with Chronic Migraine (CM) Were Sleep Disorders (70.8%), Hypo- or Hyperreflexia in Tendon Reflexes (69.8%), Cognitive Impairment (56.6%), and Emotional Lability (54.7%). These symptoms are associated with functional deficits in the central nervous system and dysregulation of the serotonergic system. Sleep disturbances contribute to frequent attacks and shorter remission periods. Reflex abnormalities reflect increased excitability or neuropathic changes in the nervous system. Visual disturbances (photopsia and scotoma) were more often observed in patients with migraine with aura and appeared as visual signals preceding attacks. Sensory disturbances (paresthesia) commonly manifested as tingling or discomfort in the face or hands. Motor function changes, in some cases, were related to headache severity, presenting as impaired coordination or sudden weakness. Cognitive deficits (reduced attention and memory) and emotional lability intensified during attacks and were closely linked to depression and anxiety.

Thus, most patients with chronic migraine develop pronounced neurophysiological changes. These changes affect not only pain perception but also personal and cognitive functioning. Therefore, comprehensive neurological assessment in these patients should include psychological status, sleep quality, and cognitive abilities.

Autonomic Nervous System Dysfunction Plays a Key Role in CM Pathogenesis. This system regulates blood circulation, respiration, sweat gland activity, and visceral organ tone. In patients with migraine, disruption of the balance between sympathetic and parasympathetic branches is a primary pathophysiological factor underlying headache. In CM patients, frequent observations included tachycardia, labile blood pressure, cold extremities, altered sweating, facial flushing or pallor, and changes in respiratory rhythm.

Data showed that 62.3% of patients had labile blood pressure and 58.5% tachycardia. These conditions are associated with increased sympathetic activity, which may exacerbate attacks under stress, fatigue, or hormonal changes. Autonomic imbalance alters cerebral blood flow dynamics. Increased sympathetic activity can cause cerebral vasospasm and hypoperfusion, triggering migraine attacks, whereas increased parasympathetic activity may lead to vasodilation and prolonged headaches. Signs such as enhanced sweat gland activity, facial flushing, and transient visual dimming were also noted, indicating excessive autonomic reactivity and disrupted homeostatic balance.

Chronic migraine is accompanied by varying degrees of central nervous system dysfunction, particularly cognitive impairment—reduced attention, short-term memory deficits, and slowed thinking—linked to altered cerebral blood flow, neurotransmitter imbalance, and chronic pain–related mental fatigue.

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Cognitive Function Assessment in CM Patients was performed using various psychometric methods, including the Mini-Cog test, Schulte tables, verbal memory tests, and the overall cognitive index. These methods allowed objective evaluation of attention, processing speed, and memory retention. Analysis revealed that all cognitive parameters in CM patients were significantly lower compared with the control group. Attention and short-term verbal memory deficits were particularly prominent, reflecting the impact of prolonged pain, stress, and central nervous system fatigue.

Compared with controls, Mini-Cog scores averaged 3.6 ± 0.08 in CM patients versus 4.75 ± 0.05 in the control group, indicating mild cognitive impairment. Schulte table performance also showed slower attention and cognitive processing (66 s vs. 54 s). Verbal memory and recall tests indicated lower scores in CM patients. The overall cognitive index was 68.2 in CM patients versus 88.9 in controls, confirming a >20% reduction in general cognitive efficiency. These findings indicate that chronic pain and neurovegetative stress result in functional decline in memory, attention, and thinking. Cognitive impairment in migraine patients is often associated with attack frequency and duration, particularly affecting the left frontal lobe and limbic system. Impaired cognitive function negatively impacts daily activities, work efficiency, and psycho-emotional stability. Memory and attention deficits also reflect alterations in neurotransmitter balance (especially acetylcholine and serotonin systems) and elevated stress hormones due to chronic pain.

Thus, cognitive deficits in CM patients are systematic and functional in nature, reflecting overall decreased brain activity and nervous system fatigue. Early detection and targeted psychoneurological interventions may help mitigate disease progression.

Cerebral Hemodynamics Assessment in CM patients was performed using transcranial Doppler ultrasonography. The study aimed to determine the functional status of cerebral circulation and evaluate changes in arterial pulsatility and elasticity during chronic disease. Measurements were taken in the internal carotid artery (ICA), middle cerebral artery (MCA), vertebral artery (VA), posterior cerebral artery (PCA), and pulsatility index (PI), with results compared to a control group.

A decrease in MCA flow velocity (60.45 ± 0.44 vs. 65.3 ± 0.24) indicated reduced perfusion in the cerebral hemispheres, potentially related to decreased cortical activity and impaired vasoregulation contributing to headache attacks. PI was slightly increased (1.21 vs. 1.19), suggesting elevated vascular tone and a vasospastic state. Overall, Doppler findings demonstrated increased vessel wall reactivity, disrupted hemodynamic balance, and the presence of vasospastic elements in CM patients.

Conclusion

The results indicate that chronic migraine is characterized by complex clinical, neurological, and cerebral hemodynamic changes. CM attacks, unlike stroke-free headache syndromes, are accompanied by central neurovascular dysfunction, autonomic reactivity, and cognitive impairment. Neurological analysis revealed frequent sleep disturbances, reflex abnormalities, cognitive deficits, emotional lability, and sensorimotor dysfunction. These findings reflect functional deficits in the central nervous system and dysregulation of serotonergic and cholinergic systems. Cognitive testing confirmed significantly reduced attention, short-term memory, recall ability, and overall cognitive index compared with controls. Continuous pain stimulation, stress, aura episodes, and neurovegetative reactions were identified as key factors exacerbating cognitive decline.

Cerebral hemodynamic analysis revealed significantly reduced blood flow velocity in the ICA, MCA, VA, and PCA, with VA being particularly affected, indicating the involvement of the posterior circulation in migraine pathogenesis. Relatively elevated PI confirms a vasospastic state and increased vascular tone.

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Chronic migraine arises from interactions between central and peripheral neurovascular mechanisms, with neurological dysfunction, cognitive decline, and hemodynamic dysregulation determining clinical severity. Comprehensive assessment combining clinical history, cognitive testing, and cerebral Doppler ultrasonography improves diagnostic accuracy. These findings support the development of individualized therapeutic strategies aimed at reducing attack frequency, prolonging remission, and improving patients' social activity and quality of life.

References

1. Goadsby P.J., Holland P.R., Martins-Oliveira M., Hoffmann J., Schankin C., Akerman S. Pathophysiology of Migraine: A Disorder of Sensory Processing. Physiological Reviews. 2017;97(2):553–622.
2. Dodick D.W. A Phase-by-Phase Review of Migraine Pathophysiology. Headache. 2018;58(Suppl 1):4–16.
3. Ashina M., Buse D.C., Ashina H., Pozo-Rosich P. Migraine: Epidemiology and Systems of Care. Lancet. 2021;397(10283):1485–1495.
4. Charles A. The Pathophysiology of Migraine: Implications for Clinical Management. Lancet Neurology. 2018;17(2):174–182.