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CLINICAL AND BIOCHEMICAL FEATURES, DIAGNOSTICS, AND DIFFERENTIAL DIAGNOSTIC CHARACTERISTICS OF BASILAR MIGRAINE.

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Relevance. Migraine is a chronic disease manifested by recurrent severe headache attacks, beginning at an early age and persisting throughout a significant part of a patient's life. It is one of the most common neurological disorders, affecting 10–15% of the world's population, and it significantly interferes with work capacity during the most productive years of life [10]. Migraine is a benign primary headache disorder and ranks second in prevalence after tension-type headache. According to WHO data from 2000, migraine ranks 19th among men and 12th among women in the list of diseases affecting work capacity worldwide, and it occupies a leading position among neurological disorders [1].

Epidemiological studies show that migraine occurs 1.5–2 times more frequently in women than in men, affecting 11–25% of women and 4–10% of men. The disease usually first manifests between the ages of 10 and 20 years, reaches maximum headache severity at 35–45 years, and migraine attacks often cease at 55–60 years. In 60–70% of cases, migraine has a hereditary nature [2,3]. According to the 3rd edition of the International Classification of Headache Disorders (ICHD-3, 2018), migraine is classified as follows:

1.Migraine.
1.1. Migraine with aura.
1.2. Migraine without aura.
1.2.1. Typical migraine with aura.
1.2.1.1. Typical migraine with aura and headache.
1.2.1.2. Typical migraine with aura without headache.
1.2.2. Migraine with brainstem aura.
1.2.3. Hemiplegic migraine.
1.2.3.1. Familial hemiplegic migraine.
1.2.3.1.1. Familial hemiplegic migraine type 1.
1.2.3.1.2. Familial hemiplegic migraine type 2.
1.2.3.1.3. Familial hemiplegic migraine type 3.
1.2.3.2. Sporadic hemiplegic migraine.
1.2.4. Retinal migraine.
1.3. Chronic migraine.
1.4. Complicated migraine.
1.4.1. Status migrainosus.
1.4.2. Persistent aura without infarction.
1.4.3. Migrainous infarction.
1.4.4. Triggered migraine with aura.
1.5. Probable migraine.
1.5.1. Probable migraine without aura.
1.5.2. Probable migraine with aura.
1.6. Episodic syndromes that may be associated with migraine.
1.6.1. Recurrent gastrointestinal disorders.
1.6.1.1. Cyclic vomiting syndrome.
1.6.1.2. Abdominal migraine.

1.6.2. Benign paroxysmal vertigo.
1.6.3. Benign paroxysmal torticollis [20].

According to modern medical data, patients with migraine may develop cognitive impairment, acute cerebrovascular pathology (migrainous stroke), as well as degenerative changes in the white matter of the brain. Neuroimaging studies indicate that the risk of migraine-associated vascular disease in women and pregnant patients increases up to 16-fold [5].

Clinical features. The main symptom of migraine is a throbbing, paroxysmal headache of moderate intensity. Headache may occur at any time of day, gradually intensify, be unilateral or bilateral, or alternate between the right and left sides. Headache is often accompanied by nausea, vomiting, chills, excessive sweating, palpitations, dry mouth, and other autonomic disturbances [7,8]. Migraine attacks may be triggered by stress, emotional overload, menstruation, insufficient or excessive sleep, physical exertion, certain foods (cocoa, chocolate, citrus fruits, fatty foods), alcohol, constipation, nitroglycerin, bright lights, noise, unpleasant odors, exacerbation of comorbid diseases, changes in weather, and atmospheric pressure fluctuations.

Basilar migraine occupies an important place among complicated forms of migraine. According to the International Classification of Headache Disorders, basilar migraine is a migraine with aura originating from the brainstem or cerebral hemispheres and is not accompanied by motor weakness [6]. Symptoms of basilar migraine include dysarthria, vertigo, tinnitus, hypoacusis, diplopia, bilateral paresthesia, decreased level of consciousness, and ataxia. In many patients, visual and sensory aura alternates with headache. A typical basilar migraine attack begins with visual disturbances (photophobia alternating with bilateral visual impairment), followed by vertigo, ataxia, dysarthria, tinnitus, short-term paresthesia in the arms and legs, severe throbbing occipital headache, nausea, and in 30% of cases, brief loss of consciousness. Basilar migraine is considered a rare form. Vertigo is systemic and may last from several minutes to several hours [10].

Among biochemical changes in migraine, the role of brain-derived neurotrophic factor (BDNF) is of particular importance. BDNF is one of the most extensively studied neurotrophic factors produced in the brain and serves as a mediator supporting neuronal survival and regeneration. In mammals, BDNF is synthesized in developing and mature neurons, as well as in platelets, astrocytes, microglia, endothelial cells, and the liver [9]. BDNF is involved not only in memory and learning processes but also in neurogenesis, differentiation, and growth of new neurons and synapses. It stimulates the growth and regeneration of damaged nerve cells and plays an important role in neuronal recovery in various pathological processes of the brain.

In trigeminal nerve endings, BDNF increases in response to CGRP stimulation, contributing to central sensitization in migraine pathogenesis. In addition, BDNF exerts neuroprotective effects by binding to the TrkB receptor, supporting progenitor nerve cells, preventing cortical neuron degeneration, and ensuring synapse formation, maintenance, and plasticity, thereby strengthening memory. BDNF also participates in oxidative processes by regulating superoxide dismutase and glutathione reductase activity. Antioxidant proteins such as sulfiredoxin and sestrins regenerate oxidized peroxides and regulate Nrf2 antioxidant transcription factors [9].

BDNF is considered a nociceptive mediator of trigeminal nerve endings. A significant decrease in platelet BDNF levels has been identified in patients with migraine, playing an important role in migraine pathophysiology. The mechanism of central sensitization is characterized by changes in activity-dependent plasticity of second-order trigeminal neurons (Lemos et al., 2010). Sutherland et al. (2014) demonstrated in experimental and clinical studies an association between migraine and the rs2049046 polymorphism (TT genotype and T allele), showing that the presence of AT (rs2049046, BDNF) and GC (rs1553005, CGRP) genotypes increases the risk of

migraine attacks. A study conducted in Brazil by Tanure et al. in 2010 showed a significant increase in serum BDNF levels during migraine attacks in patients with migraine.

Diagnostics. Diagnostic criteria for basilar migraine:

A. Criteria B–G must be met, and migraine attacks must have occurred at least twice.

B. At least two of the following fully reversible symptoms must be present:

1. Dysarthria.
2. Vertigo.
3. Tinnitus.
4. Hypoacusis.
5. Diplopia.
6. Visual impairment (nasal or temporal hemianopia).
7. Ataxia.
8. Impaired consciousness.
9. Bilateral paresthesia.

C. At least one of the following must be present:

10. At least one aura symptom develops gradually over not less than 5 minutes.
11. Each symptom lasts not less than 5 minutes and not more than 60 minutes.

D. Headache meets criteria B–G.

E. Headache is not better explained by another disorder [4].

Differential diagnosis. Basilar migraine should be differentiated from Ménière's disease and cervical migraine (Barre–Lieou syndrome).

Differential diagnosis of Ménière's disease and basilar migraine is based on clinical and diagnostic features [4,6,7].

Characteristics of attacks	**Ménière's disease attacks**	**Basilar migraine**
Vertigo	Systemic	Non-systemic
Tinnitus	Typical	May be present
Hearing loss	Typical	Atypical
Coordination impairment	Typical	Typical
Visual disturbances	Atypical	Typical
Loss of consciousness	Not typical	Observed
Nausea, vomiting	Repeated	At the peak of pain
Duration	From 20 minutes to 3 hours	Up to 1 hour
Headache	Not typical	Occurs during aura
Relief of attack	Atropine, scopolamine, diazepam	NSAIDs, calcium channel blockers

Symptom / Feature	**Meniere's Disease Attacks**	**Basilar Migraine Attacks**
Vertigo	Systemic	Non-systemic
Tinnitus (ear noise)	Typical	May occur
Hearing loss	Typical	Atypical
Coordination disturbance	Typical	Typical
Visual disturbance	Atypical	Typical
Fainting / Loss of	—	Observed

Symptom / Feature	Meniere's Disease Attacks	Basilar Migraine Attacks
consciousness		
Nausea, vomiting	Repeated episodes	During severe attacks
Duration	20 minutes to 3 hours	Up to 1 hour
Headache	—	Observed during aura
Treatment / Relief of attack	Atropine, scopolamine, diazepam	Triptans, calcium channel blockers

Differential features of cervical migraine and basilar migraine [3,4,5].

Clinical Feature	Cervical Migraine	Basilar Migraine
Triad neurological symptoms	Appear simultaneously with headache or at any time during pain	Appear 1 hour before headache phase, may persist during pain
Syncope	Not typical	Typical
Headache character	Throbbing or shooting	Pulsating or crushing
Headache localization	Spreads from neck to one half of head	Occipital areas
VAS headache intensity	Up to 10 points	Up to 5–6 points
Photophobia	Rare	Frequent
Phonophobia	Rare	Frequent
Scleral hyperemia, lacrimation	Typical	Not typical
Attack duration	30 seconds to 24 hours	More than 24 hours
Abortive treatment	Triptans, novocaine blockade	Triptans, calcium channel blockers
Neck muscle tension	Pronounced	May occur
Neck muscle trigger points	Present	Not typical

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Conclusion:

1. The BDNF (Brain-Derived Neurotrophic Factor) gene plays a crucial role in the development of migraine.
2. Basilar migraine has stronger and more specific clinical and diagnostic features compared to other types of migraine.
3. In patients with basilar migraine, accurate and timely diagnosis can prevent the progression of migraine into complications or chronic forms.
4. Patients with basilar migraine require long-term individualized diagnostics and a rehabilitation program based on pathogenetic mechanisms.

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