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Diagnosis of the Pathogenesis of Migraine Disease and Modern Clinical Diagnostic Methods

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Abstract: A type of migraine headache. Typically, the attack lasts from 4 to 72 hours and symptoms can be severe. The pain is often unilateral, throbbing, aggravated by physical activity, and may be accompanied by symptoms such as nausea, sensitivity to light, sound, and odors. In 25% of patients, precursors of epileptic seizures often develop before, but sometimes after, the headache attack. The diagnosis is made on the basis of clinical data. Treatment includes triptans, dihydroergotamine, antiemetics, and analgesics. Prevention includes lifestyle changes (e.g., sleep or diet) and medications (e.g., beta-blockers, amitriptyline, topiramate, divalproex, monoclonal antibodies).

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Migraine epidemiology

Migraine is the most common cause of recurrent headaches of moderate to severe intensity; in the United States, the prevalence of this pathology over 1 year is 18% in women and 6% in men. Migraine usually begins at a young age and then its intensity varies over the years; after 50 years, the disease often regresses. Some studies have shown a hereditary predisposition to the development of migraine.

Based on survey data among veterans of the conflicts in Iraq and Afghanistan, it has been suggested that migraines may develop after head injuries.

Pathophysiology of migraine

Migraine is a neurovascular headache syndrome in which there is a change in the functional activation of the central nervous system (activation of brain nuclei, hyperexcitability, then the spread of depression of the cerebral cortex) and the involvement of the trigeminal neurovascular complex. nerve in the process (the release of neuropeptides leads to painful vascular inflammation of the skull and dura mater).

Many factors have been identified that can trigger a migraine attack, such as:

a) Drinking red wine

b) Malnutrition

c) Strong irritants (e.g., flashing lights, strong odors)

d) Weather changes

e) Insomnia

f) Stress

g) Hormonal factors, especially related to menstruation

h) Some foods

i) Refreshing foods vary from person to person.

In some cases, a migraine attack or migraine exacerbation can be caused by a head injury, neck pain, or temporomandibular joint dysfunction.

Changes in estrogen levels are also a possible trigger. In many patients, the onset of migraines coincides with the menstrual cycle, with severe attacks developing during menstruation (called menstrual migraines), and worsening during menopause. Many women experience remission of migraines during pregnancy (although in some cases they worsen in the first or second trimester of pregnancy); they worsen after the baby is born, when estrogen levels drop sharply.

Taking oral contraceptives and other hormonal medications sometimes triggers or worsens migraine attacks and is also a risk factor for stroke in women with migraine with aura.

Familial hemiplegic migraine is a rare subtype of migraine that is associated with defects in genes located on chromosomes 1, 2, and 19. The role of a genetic component in the more common forms of migraine is currently being studied. In some families, the manifestations of migraine are very diverse, with some family members having headaches and others having dizziness, hemiplegia, or aura. These findings suggest that migraine may be a more general disorder than just headaches.

Migraine signs and symptoms

Attacks are often preceded by a prodrome (a feeling that a migraine is starting). The prodrome may include mood swings, neck pain, cravings for certain foods, loss of appetite, nausea, or a combination of these symptoms.

About 25% of patients experience warning signs before attacks. Auras are temporary neurological disorders that can affect sensation, balance, coordination, speech, or vision; they last from a few minutes to an hour. The aura may persist after the onset of the headache episode. Often, visual disturbances develop with auras (flashes of light, arcs of flashing lights, bright zigzags, scotomas). Less common are paresthesias and numbness (usually starting in the hand, then moving to the arm and face), speech disorders, and temporary brainstem dysfunction (manifested as ataxia, confusion, or even obtundation). In some patients, the aura develops with little or no headache.

The intensity of the headache varies from moderate to severe, attacks last from 4 hours to several days, usually disappearing after sleep. Most often, the pain is unilateral, but it can spread to both halves, is pulsating in nature, the most common localization is the frontotemporal region.

However, migraine is not just a headache. It is accompanied by a number of symptoms, such as nausea (sometimes vomiting), photophobia, phonophobia and osmophobia. During an attack, patients complain of difficulty concentrating. Ordinary physical activity usually increases the intensity of the headache; therefore, also due to the development of photo and phonophobia, patients prefer to be in a dark room during an attack, with a minimum of extraneous sounds. In severe cases, attacks are practically disabling for patients, preventing them from working and disrupting family life.

The frequency and intensity of attacks vary greatly. Many patients experience several types of headaches, including milder attacks that are not accompanied by nausea or photophobia; they may resemble tension headaches in clinical presentation, but are actually a form of migraine.

Chronic migraine

Patients with episodic migraine may later develop chronic migraine. In such patients, headache attacks occur ≥ 15 times per month. Previously, this pathology was called mixed or combined headache, because it had features of migraine and tension headache. Such headaches often occur in patients who use drugs to relieve headache attacks.

Other symptoms

In other, rarer forms of migraine, other symptoms may develop:

Migraine with brainstem aura (formerly called basilar migraine) causes a combination of dizziness, ataxia, visual field loss, sensory disturbances, focal weakness, and changes in level of consciousness.

In the hemiplegic form of migraine (which can be sporadic or familial), unilateral paresis develops.

Migraine diagnosis

a) Clinical assessment

b) When diagnosing migraine, they rely on characteristic symptoms and the absence of pathology during an objective examination, including a detailed assessment of the neurological status.

c) Red flags that may indicate the presence of another pathology (even in patients with migraines) include:

d) Headache that reaches peak intensity within a few seconds (thunderclap type)

e) Over 50 debuts

f) Headache attacks that are severe and/or frequent for several weeks or more

g) History of malignant tumor (brain metastases) or immunodeficiency disorder (e.g., HIV infection, AIDS)

h) Fever, meningism, altered consciousness, or a combination of these

i) Persistent focal neurological symptoms

j) Papillary tumor

k) A clear change in the nature of the headache

Patients with a characteristic clinical presentation and no warning signs listed above do not require further investigation. If red flags are present, tests are often needed, including MRI and in some cases, lumbar puncture.

The most common diagnostic errors include:

a) Although migraines can cause bilateral pain, it is not always described as throbbing.

b) Misdiagnosis of migraine as a migraine headache or eye strain occurs due to the absence of autonomic and visual symptoms of migraine.

c) Any episode of headache in a patient with confirmed migraine represents a migraine attack (a thunderclap headache or a change in the pattern of a typical headache may indicate a new, potentially serious pathology)

d) Migraine with aura may be called a transient ischemic attack, especially if the aura occurs without headache in older people;

e) Diagnosing a sudden headache as a migraine because it goes away after taking a triptan (taking triptans also relieves headaches in subarachnoid hemorrhage)

f) Some rare diseases can mimic migraine with aura:

g) Carotid or vertebral artery dissection

h) Cerebral vasculitis

i) Moyamoya disease

j) CADASIL (cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy)

k) MELAS syndrome (mitochondrial encephalomyopathy, lactic acidosis, and stroke-like attacks)

Migraine treatment

a) Eliminate Specific Triggers

b) Relaxation techniques, yoga, or behavioral interventions

c) For mild attacks, use acetaminophen or nonsteroidal anti-inflammatory drugs (NSAIDs).

d) In acute attacks, it is recommended to take triptans, lasmiditan, gepants (calcitonin gene-dependent peptide antagonists [ubrogepant, etc.]) or dihydroergotamine together with an antiemetic - a dopamine antagonist.

e) Neuromodulatory devices for acute treatment and prevention

Providing patients with a detailed explanation of the characteristics of a disease such as migraine helps them understand that although this pathology cannot be cured, it can be controlled, which encourages patients to participate more actively in their treatment.

The patient is advised to keep a headache diary, recording the number and time of attacks, triggers, and response to medication. If possible, identified triggers are eliminated. However, eliminating triggers may be redundant.

The choice of medications for acute migraine depends on the frequency, duration, and severity of attacks. Analgesics, antiemetics, triptans, lasmiditan, gepanthate (small molecule calcitonin gene-related peptide [CGRP] receptor antagonists), or dihydroergotamine (1) may be used. If patients wish to avoid drug therapy or if drug therapy is ineffective, neuromodulatory therapy may sometimes be used to control and/or prevent acute attacks.

Patients who frequently (e.g., more than 2 days per week) use medications (especially analgesics containing butalbital, triptans, ergotamine, or opioids) to treat acute migraine attacks should be treated with migraine preventive medications in conjunction with a program to stop excessive painkiller use.

Clinicians sometimes recommend the use of behavioral psychotherapy (biofeedback, stress management, psychotherapy) in cases where stress is the main cause of headaches, as well as in cases of excessive use of analgesics.

Yoga can reduce the frequency and intensity of headaches; it increases vagal tone and decreases sympathetic tone, thereby improving autonomic balance of the heart. Relaxation techniques can reduce sympathetic nervous system activity, reduce muscle tension, and alter brain wave activity.

Acute attacks

NSAIDs or acetaminophen are prescribed for mild to moderate migraine attacks.

If these medications are ineffective, triptans or dihydroergotamine should be considered. Effective headache relief with triptans or dihydroergotamine should not be considered diagnostic criteria for migraine, as these same medications may relieve pain due to subarachnoid hemorrhage or other organic brain lesions.

If mild attacks become severe or attacks are severe at the onset, triptans or dihydroergotamine may be used. For severe nausea, a combination of a triptan with an antiemetic, also taken at the beginning of the attack, is effective.

They are selective agonists of serotonin 1B and 1D receptors. Although not specific analgesics, triptans specifically block the release of neuropeptides that trigger a headache attack. These drugs are most effective when taken at the beginning of an attack. They are available in oral, intranasal, and subcutaneous forms; the subcutaneous forms are more effective but have more side effects. Overuse of triptans can also lead to the development of headaches. Taking triptans and dihydroergotamine can provoke spasm of the coronary arteries, so these drugs are contraindicated in patients with coronary heart disease or malignant arterial hypertension; they should be used with caution in elderly patients

and in the presence of risk factors for vascular pathology. Alternatives are ubrogepant and rimegepant, which are hepants.

If triptans or dihydroergotamine are contraindicated due to cardiovascular disease, lasmiditan (a new selective serotonin [5-HT] 1F receptor agonist) or a gepant such as ubrogepant or rimegepant may be prescribed. Lasmiditan, which has a much greater affinity for serotonin 1F receptors than for 1B receptors, has no cardiovascular contraindications. (Triptans cause vasoconstriction by activating 5-HT1B receptors.) There are currently no specific cardiovascular precautions or contraindications for the use of gepant, and no serious cardiovascular or gastrointestinal side effects are known.

Antiemetics (e.g., metoclopramide, prochlorperazine) used as monotherapy can relieve mild to moderate headaches. If you cannot tolerate triptans and other vasoconstrictors, you can use prochlorperazine in the form of suppositories (25 mg) or tablets (10 mg).

There is evidence to support the use of neuromodulators in the treatment of acute attacks and the prevention of chronic migraine.

Non-stop seizures

Intravenous administration of large volumes of fluid (e.g., 1-2 liters of 0.9% saline solution) can help relieve headache and improve well-being, especially in patients who are dehydrated due to vomiting.

For severe, prolonged attacks, intravenous dihydroergotamine in combination with a dopamine receptor antagonist (e.g., metoclopramide 10 mg intravenously, prochlorperazine 5-10 mg intravenously) is effective. Dihydroergotamine is also available in intranasal and subcutaneous forms.

Opioids should only be used as a last resort when other treatments have failed.

Chronic migraine

Chronic migraine is treated with the same medications used for episodic migraine, including monoclonal antibodies that block CGRP. There is also strong evidence in favor of onabotulinumtoxinA and topiramate.

Evidence supports the use of neurostimulation for the treatment and prevention of attacks in chronic migraines. Noninvasive options include supraorbital stimulation, vagus nerve stimulation, monopulse transcranial magnetic stimulation, and remote electrical stimulation.

Neuromodulator treatment

Neuromodulation treatments, which affect brain activity using electrical currents or magnetic fields, can be non-invasive with commercially available devices. Such techniques are also used to treat and prevent seizures.

Noninvasive transcranial magnetic stimulation, using a handheld device placed on the back of the head, may relieve acute migraines (3). A device that uses a patch that delivers painless electrical stimulation to the skin (called remote electrical neuromodulation) may relieve acute migraine pain. Using a portable device that provides noninvasive stimulation of the vagus nerve is also effective.

Trigeminal nerve stimulation using a forehead-mounted device can be used to treat acute migraine attacks (with or without aura) or to reduce the frequency of attacks in patients ≥ 18 years of age.

Non-invasive neuromodulation devices are not associated with significant side effects. Invasive treatments are usually only available in specialized centers and carry more risks than non-invasive treatments.

Prognosis for migraine

For some patients, migraine attacks are infrequent and very tolerable. For others, migraine is a debilitating condition, causing frequent episodes of disability, loss of ability to perform productive activities, and a significant reduction in quality of life.

Migraine prevention

When frequent migraines interfere with daily activities despite acute treatment, daily preventive treatment is warranted. Some experts consider onabotulinumtoxinA to be the drug of choice.

For patients with frequent analgesic use (e.g., more than twice a week), especially those with medication overuse headaches, preventive treatment should be combined with interventions to stop analgesic overuse. The choice of medication may be based on co-morbidity

Amitriptyline before bed for patients with insomnia

Beta-blockers - in patients with anxiety or coronary artery disease

Topiramate to help with weight loss - in obese patients or those who do not want to gain weight

Monoclonal antibodies (e.g., erenumab, fremanezumab, galcanezumab), when other drugs have shown ineffectiveness

Gepant can be used for acute attacks (ubrogepant, rimegepant) and for migraine prevention (atogepant, rimegepant).

List of used literature:

1. Marmura MJ, Silberstein SD, Schwedt TJ: Acute treatment of migraine in adults: an American Headache Society assessment of migraine pharmacotherapies. Headache 55(1):3-20, 2015.
2. Miller S, Sinclair AJ, Davies B, Matharu M: Neurostimulation in the treatment of primary headache. Pract Neurol 16(5):362–375, 2016. doi:10.1136/pactneurol-2015-001298
3. Lipton RB, Dodick DW, Silberstein SD, et al.: Single-pulse transcranial magnetic stimulation for the acute treatment of migraine with aura: A randomized, double-blind, parallel-group, sham-controlled trial. Lancet Neurol 9:373–380, 2010. doi: 10.1016/S1474-4422(10)70054-5
4. Andryev S. et al. Experience with the use of memantine in the treatment of cognitive disorders //Science and innovation. – 2023. – T. 2. – №. D11. – C. 282-288.
5. Antsiborov S. et al. Association of dopaminergic receptors of peripheral blood lymphocytes with a risk of developing antipsychotic extrapyramidal diseases //Science and innovation. – 2023. – T. 2. – №. D11. – C. 29-35.
6. Asanova R. et al. Features of the treatment of patients with mental disorders and cardiovascular pathology //Science and innovation. – 2023. – T. 2. – №. D12. – C. 545-550.
7. Begbudiyev M. et al. Integration of psychiatric care into primary care //Science and innovation. – 2023. – T. 2. – №. D12. – C. 551-557.
8. Bo'Riyev B. et al. Features of clinical and psychopathological examination of young children //Science and innovation. – 2023. – T. 2. – №. D12. – C. 558-563.
9. Borisova Y. et al. Concomitant mental disorders and social functioning of adults with high-functioning autism/asperger syndrome //Science and innovation. – 2023. – T. 2. – №. D11. – C. 36-41.
10. Ivanovich U. A. et al. Efficacy and tolerance of pharmacotherapy with antidepressants in non-psychotic depressions in combination with chronic brain ischemia //Science and Innovation. – 2023. – T. 2. – №. 12. – C. 409-414.
11. Nikolaevich R. A. et al. Comparative effectiveness of treatment of somatoform diseases in psychotherapeutic practice //Science and Innovation. – 2023. – T. 2. – №. 12. – C. 898-903.
12. Novikov A. et al. Alcohol dependence and manifestation of autoagressive behavior in patients of different types //Science and innovation. – 2023. – T. 2. – №. D11. – C. 413-419.

13. Pachulia Y. et al. Assessment of the effect of psychopathic disorders on the dynamics of withdrawal syndrome in synthetic cannabinoid addiction //Science and innovation. – 2023. – T. 2. – №. D12. – C. 240-244.
14. Pachulia Y. et al. Neurobiological indicators of clinical status and prognosis of therapeutic response in patients with paroxysmal schizophrenia //Science and innovation. – 2023. – T. 2. – №. D12. – C. 385-391.
15. Pogosov A. et al. Multidisciplinary approach to the rehabilitation of patients with somatized personality development //Science and innovation. – 2023. – T. 2. – №. D12. – C. 245-251.
16. Pogosov A. et al. Rational choice of pharmacotherapy for senile dementia //Science and innovation. – 2023. – T. 2. – №. D12. – C. 230-235.
17. Pogosov S. et al. Gnostic disorders and their compensation in neuropsychological syndrome of vascular cognitive disorders in old age //Science and innovation. – 2023. – T. 2. – №. D12. – C. 258-264.
18. Pogosov S. et al. Prevention of adolescent drug abuse and prevention of yatrogenia during prophylaxis //Science and innovation. – 2023. – T. 2. – №. D12. – C. 392-397.
19. Pogosov S. et al. Psychogenetic properties of drug patients as risk factors for the formation of addiction //Science and innovation. – 2023. – T. 2. – №. D12. – C. 186-191.
20. Prostyakova N. et al. Changes in the postpsychotic period after acute polymorphic disorder //Science and innovation. – 2023. – T. 2. – №. D12. – C. 356-360.
21. Prostyakova N. et al. Issues of professional ethics in the treatment and management of patients with late dementia //Science and innovation. – 2023. – T. 2. – №. D12. – C. 158-165.
22. Prostyakova N. et al. Sadness and loss reactions as a risk of forming a relationship together //Science and innovation. – 2023. – T. 2. – №. D12. – C. 252-257.
23. Prostyakova N. et al. Strategy for early diagnosis with cardiovascular diseaseisomatized mental disorders //Science and innovation. – 2023. – T. 2. – №. D12. – C. 166-172.
24. Rotanov A. et al. Comparative effectiveness of treatment of somatoform diseases in psychotherapeutic practice //Science and innovation. – 2023. – T. 2. – №. D12. – C. 267-272.
25. Rotanov A. et al. Diagnosis of depressive and suicidal spectrum disorders in students of a secondary special education institution //Science and innovation. – 2023. – T. 2. – №. D11. – C. 309-315.
26. Rotanov A. et al. Elderly epilepsy: neurophysiological aspects of non-psychotic mental disorders //Science and innovation. – 2023. – T. 2. – №. D12. – C. 192-197.
27. Rotanov A. et al. Social, socio-cultural and behavioral risk factors for the spread of hiv infection //Science and innovation. – 2023. – T. 2. – №. D11. – C. 49-55.
28. Rotanov A. et al. Suicide and epidemiology and risk factors in oncological diseases //Science and innovation. – 2023. – T. 2. – №. D12. – C. 398-403.
29. Sedenkov V. et al. Clinical and socio-demographic characteristics of elderly patients with suicide attempts //Science and innovation. – 2023. – T. 2. – №. D12. – C. 273-277.
30. Sedenkov V. et al. Modern methods of diagnosing depressive disorders in neurotic and affective disorders //Science and innovation. – 2023. – T. 2. – №. D12. – C. 361-366.