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EARLY DIAGNOSIS OF CENTRAL NERVOUS SYSTEM DISEASES AND THEIR COMPLICATIONS

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Abstract: The most common diseases of the nervous system are acute pathologies affecting the blood vessels of the brain. These include stroke, dyscirculatory encephalopathy, intracerebral hemorrhages, intracranial arterial aneurysms; Consequences of traumatic brain injuries; Infectious diseases of the central nervous system: meningitis, encephalitis, brain abscess, poliomyelitis; Multiple sclerosis; Epilepsy; Age-related diseases of the nervous system - Parkinson's disease and Alzheimer's disease; Osteocondritis of the spine; Genetic and inherited diseases of the nervous system.

Keywords: epilepsy,age-related diseases,Parkinson's disease, Alzheimer's disease, nervous system, dyslipidemia, homeostasis.

The human nervous system is anatomically divided into two parts: central (CNS) and peripheral (PNS). This complex structure permeates all the organs and tissues of our body. It is thanks to it that self-regulation of all vital functions of the body is possible, as well as the perception, storage and processing of received information. Nerve impulses allow us to sense and perceive the world around us in all its diversity. However, due to the complexity and fragility of the structure of the nervous system, injuries, damage and diseases (including genetic ones) can disrupt its coordinated work. The most common diseases of the nervous system are acute pathologies affecting the blood vessels of the brain. These include stroke, dyscirculatory encephalopathy, intracerebral hemorrhages, intracranial arterial aneurysms; Consequences of traumatic brain injuries; Infectious diseases of the central nervous system: meningitis, encephalitis, brain abscess, poliomyelitis; Multiple sclerosis; Epilepsy; Age-related diseases of the nervous system - Parkinson's disease and Alzheimer's disease; Osteocondritis of the spine; Genetic and inherited diseases of the nervous system. This group usually includes pathologies such as Leigh's syndrome, Tourette's syndrome,

spinal muscular atrophy, Huntington's and Batten's diseases. Various disorders in the functioning of the peripheral nervous system: neuralgia, neuritis, lumbodynia and others. Ischemic stroke is a sudden neurological failure caused by focal cerebral ischemia with permanent cerebral infarction (eg, positive findings on diffusion-weighted MRI). The most common causes are: atherothrombotic occlusions of large arteries; embolism of cerebral vessels (embolic infarction); non-thrombotic occlusion of small, deep cerebral arteries (lacunar infarction); and proximal arterial stenosis with hypotension, which reduces cerebral circulation in arterial divide zones (hemodynamic stroke). In a third of cases of ischemic strokes, by the time the patient is discharged from the hospital, the cause has not been established; such strokes are classified as cryptogenic. The diagnosis is made based on clinical data, but CT or MRI is performed to exclude hemorrhage and confirm the presence of a heart attack and its size. In some patients, thrombolytic therapy is effective in the acute period. Depending on the cause of the stroke, carotid endarterectomy or stenting, and the use of antiplatelet or anticoagulant medications may help reduce the risk of subsequent strokes.

The following modifiable risk factors contribute to an increased risk of ischemic stroke:

Arterial hypertension

Smoking

Dyslipidemia

Diabetes

Insulin resistance

Abdominal obesity

Obstructive sleep apnea syndrome

Alcohol abuse

Lack of physical activity

High-risk diet (eg, high in saturated fat, trans fat, and calories)

Psychosocial stress (eg, depression)

Cardiac disorders (in particular, disorders that predispose to the development of embolism, such as acute myocardial infarction, infective endocarditis and atrial fibrillation)

Carotid artery stenosis

Use of certain drugs (eg, cocaine, amphetamine)

Hypercoagulation

Vasculitis

Use of exogenous estrogen

Unavoidable risk factors include the following:

History of stroke

Floor

Race/Ethnicity

Elderly age

Family history of stroke

The most common causes of ischemic stroke can be classified as

Cryptogenic stroke (ie, no obvious cardio embolic, lacunar, or atherosclerotic cause; most common classification)

Cardioembolism

Lacunar infarctions

Large vessel atherosclerosis (4th most common cause)

Cryptogenic stroke

A stroke is classified as cryptogenic if one of the following is present:

Diagnostic evaluation is incomplete.

Despite a comprehensive examination, the cause has not been established.

There is more than one likely cause (eg, atrial fibrillation and ipsilateral carotid artery stenosis).

Embolic stroke of unknown source (ESUS), a subcategory of cryptogenic stroke, is diagnosed when the source has not been identified after sufficient diagnostic evaluation has excluded lacunar stroke, underlying cardioembolic sources, and ipsilateral stenosis-occlusive disease (>50% occlusion). Recent evidence suggests that symptomatic nonstenotic carotid artery disease with <50% occlusion may be an important cause of stroke

Cardioembolism

An embolus can block any part of the arterial system.

Emboli can form as blood clots in the cavities of the heart, especially in the presence of the following factors:

Atrial fibrillation

Rheumatic heart disease (usually mitral valve stenosis)

After myocardial infarction

Vegetations on heart valves with bacterial or toxic endocarditis

Atrial myxoma

With prosthetic heart valves

Mechanical circulatory assist devices

Other sources of embolism may be blood clots formed during open heart surgery, and atheromas of extracranial vessels - the aortic arch and neck vessels. Less commonly, embolism can be fat (with fractures of long bones), gas (with decompression sickness), and also embolism with venous thrombi that pass from the right half of the heart to the left through an open oval window (paradoxical embolism). Emboli break off spontaneously or after invasive manipulations of the heart and blood vessels (for example, during catheterization). In rare cases, embolic stroke develops as a

result of thrombosis of the subclavian artery, and in this case the vertebral arteries and their branches are exposed to embolism.

Lacunar infarctions

Ischemic stroke may also be associated with the formation of lacunar infarcts. These small (≤ 1.5 cm) infarcts are caused by obstruction of small perforating arteries that supply deep cortical structures. It is believed that the cause of obstruction of these vessels is lipohyalinosis (degeneration of the wall of small arteries and its replacement with lipids and collagen). Lacunar infarctions can be caused by emboli. Lacunar strokes >1.5 cm in patients without cardiovascular risk factors (eg, hypertension, diabetes, smoking) suggest a central source of embolism. Lacunar infarctions occur more often in patients with diabetes mellitus or inadequately controlled hypertension.

Atherosclerosis of large vessels

Large vessel atherosclerosis can affect intracranial or extracranial arteries.

An atherosclerotic plaque, especially an ulcerated one, is a source of thrombus formation. The formation of an atherosclerotic plaque is possible in any of the large cerebral arteries, but is more common in areas of turbulent blood flow, especially in the area of the carotid artery bifurcation. Most often, incomplete thrombosis or thrombotic occlusion is formed in the main trunk of the middle cerebral artery and its branches, as well as large arteries of the base of the brain, deep perforating arteries and small cortical branches.

The basilar artery and the supraclinoid portion of the internal carotid artery are often affected, i.e., its segment between the cavernous sinus and the supraclinoid process.

Any factor that impairs the circulatory system (eg, carbon monoxide toxicity, severe anemia or hypoxia, polycythemia, hypotension) increases the risk of developing all types of ischemic strokes. Stroke can occur in border areas, between the bloodstreams of individual arteries; in such areas the blood supply is normally low, especially if patients are hypotensive and/or if the main cerebral arteries are narrowed.

Less commonly, ischemic stroke develops due to vascular spasm (eg, migraine, after subarachnoid hemorrhage, use of sympathomimetic drugs or drugs such as cocaine or amphetamines) or thrombosis of the venous sinuses (eg, intracranial infection, after surgery, childbirth, secondary hypercoagulability).

Insufficient blood flow in a single cerebral artery can often be compensated by the efficient functioning of the collateral system, especially between the carotid and vertebral arteries through anastomoses in the circle of Willis, and, to a lesser extent, between the large arteries of the cerebral hemispheres. However, anatomical variations in the circle of Willis and collateral vessel diameter, atherosclerosis, and other acquired arterial lesions can interrupt collateral flow, increasing the likelihood that occlusion of a single artery will cause cerebral ischemia.

Some neurons die when cerebral perfusion is reduced to <5% of normal for >5 minutes, while

the size of the lesion depends on the severity of ischemia. With mild ischemia, the process of damage to nervous tissue proceeds slowly. Thus, if perfusion decreases by 40% of normal values, then it may take 3–6 hours for all neurons in the ischemic zone to die. If severe ischemia continues for > 15–30 minutes, all affected tissues die (infarction).

Damage develops more quickly under hyperthermia and more slowly under hypothermia. If tissues are in a state of ischemia, but the damage is still reversible, then rapid resumption of blood flow can prevent tissue necrosis or reduce its volume. For example, interventions have the ability to restore the viability of moderately ischemic tissue (penumbra), which is often surrounded by areas of severe ischemia; penumbra exist thanks to collateral blood flows.

Mechanisms of ischemic injury include the following:

Edema

Microvascular thrombosis

Programmed cell death (apoptosis)

Infarction with cell necrosis

Inflammatory mediators (eg, interleukin-1-beta, tumor necrosis factor-alpha) contribute to the occurrence of microvascular edema and thrombosis. Swelling, if acute or extensive, can increase intracranial pressure.

Many factors can lead to cell death: a decrease in adenosine triphosphate (ATP) reserves, disruption of ionic homeostasis (including the accumulation of intracellular calcium), lipid peroxidation by free radicals with damage to cell membranes (iron-mediated process), the action of excitatory neurotoxins (for example, glutamate) and intracellular acidosis due to lactate accumulation.

Symptoms and signs of ischemic stroke depend on the area of the brain affected. The clinical picture often makes it possible to determine which artery is affected (see table Selected stroke syndromes), but, as a rule, there is no complete correspondence.

The most severe neurological deficit can develop within a few minutes, usually in the case of embolism. Less commonly, failure develops slowly, usually over 24–48 hours (called a “developmental stroke”), usually in an atherothrombotic stroke.

In most of these strokes, unilateral neurological symptoms (often starting in one arm and spreading ipsilaterally) progress without headache, fever, or pain in the affected parts of the body. The progression of symptoms is usually stepwise, alternating with periods of stabilization.

A stroke is considered subtotal if there is residual function in the affected area, which implies the presence of viable tissue in the area at risk of damage.

Embolic strokes occur more often during the daytime, and the onset of neurological symptoms is often preceded by headache. Blood clots usually form at night and are therefore first noticed upon awakening.

Lacunar infarcts may result in one of the classic lacunar syndromes (eg, pure motor hemiparesis, pure sensory hemianesthesia, combined hemiparesis and hemianesthesia, ataxic hemiparesis, dysarthria and clumsy hand syndrome); There are no signs of cortical dysfunction (eg, aphasia). The consequence of repeated lacunar infarctions may be the development of post-infarction dementia.

When a stroke occurs, a seizure may occur, much more often with an embolic than with a thrombotic stroke. Seizures may also occur months or years later; late seizures result from scarring or hemosiderin deposition at the site of ischemia.

Sometimes there is a fever.

The increase in neurological deficit, in particular impairment of consciousness during the first 48–72 hours, is most often due to increasing cerebral edema, but may also be associated with an expansion of the infarction zone. If the infarction is small, then functional improvement is noticeable already in the first days of the disease; further recovery occurs more gradually up to 1 year.

Initial clinical examination

Neuroimaging and determination of blood glucose levels using test strips

Testing to determine the cause of stroke

The diagnosis of ischemic stroke should be assumed when there is a sudden onset of neurological disorders corresponding to the blood supply zone of one of the cerebral arteries. Ischemic stroke must be distinguished from other causes of similar focal deficits (sometimes called stroke mimics, which are non-cerebrovascular diseases that cause focal neurological signs (eg, hypoglycemia), such as

Seizures (eg, with postictal palsy)

Infectious lesion of the central nervous system

Functional neurological disorders (usually diagnosed by exclusion)

Migraine (eg, hemiplegic migraine)

Headache, coma or stupor, and vomiting are more likely with a hemorrhagic stroke than with an ischemic stroke.

When a stroke is suspected, doctors can use standardized criteria to assess severity and track changes over time. This approach may be particularly useful as an outcome measure in effectiveness studies. The National Institutes of Health Stroke Scale (NIHSS) is a 15-point scale that assesses a patient's level of consciousness, language function, and motor and sensory impairment by asking the patient to answer questions and perform physical and mental tasks.

The evaluation of a patient with ischemic stroke requires assessment of the brain parenchyma, vasculature (including the heart and major arteries), and blood.

Differentiating different types of stroke based on clinical features is imprecise; however, some guidelines based on the progression of symptoms, time of onset, and type of disorder may help make

the diagnosis.

Although a preliminary diagnosis is made by clinical symptoms, neuroimaging and determination of blood glucose levels using test strips are urgent measures.

Clinical distinctions between lacunar, embolic, and thrombotic stroke based on history, examination, and neuroimaging are not always reliable, so additional testing is routinely performed to identify common or treatable causes and risk factors for stroke. Patients should be screened for the following categories of causes and risk factors:

Cardiac (eg, atrial fibrillation, potential structural features of embolism)

Vascular (eg, critical arterial stenosis identified on vascular imaging)

Blood disorders (eg, diabetes, dyslipidemia, hypercoagulability)

With cryptogenic strokes, the cause cannot be identified.

Brain assessment

Neuroimaging with computed tomography or magnetic resonance imaging is performed primarily to rule out intracerebral hemorrhage, subdural or epidural hematoma, or a rapidly growing, bleeding, or suddenly symptomatic tumor. Signs of computed tomography even with a large ischemic stroke in the carotid artery may be insignificant during the first few hours. Changes may include effacement of the sulci or insular cortical arch, loss of the gray-white junction between the cortex and white matter, and evidence of increased middle cerebral artery density.

Within 6-12 hours of ischemia, medium- and large-sized infarcts become visible as areas of reduced density. Small infarcts (for example, lacunar) can only be detected with magnetic resonance imaging.

Diffusion-weighted magnetic resonance imaging (a highly sensitive test for early ischemia) can be performed immediately after the initial CT scan.

Pathology of the cardiovascular system

Analysis of cardiac parameters typically includes electrocardiography, telemetry or Holter monitoring, serum troponin levels, and transthoracic or transesophageal echocardiography. Implantable cardiac monitors are useful in identifying primary atrial arrhythmias in cryptogenic stroke patients Vascular diseases

Analysis of vascular parameters may include magnetic resonance angiography (MRA), CT angiography (CTA), bilateral carotid and transcranial duplex ultrasonography, and conventional angiography. The choice and sequence of examination testing is individual, based on clinical data. MRA, CT-A and carotid ultrasonography equally well show the circulation of the anterior part of the brain (carotid basin); however, MRA and CT-A provide better images of the posterior brain circulation than carotid ultrasonography of the carotid artery. In general, CTA is preferable to MRA because motion artifacts are avoided. In general, CTA or MRA should be performed urgently, but treatment with IV tissue plasminogen activator (tPA) should not be delayed if indicated.

Blood-related causes

To identify causes related to blood disorders (eg, thrombotic pathologies), blood tests are done to evaluate the contribution of these pathologies and the contribution of other causes. Routine testing typically includes a full blood count (CBC), blood chemistry panel, prothrombin time/partial thromboplastin time (PT/PTT), fasting blood glucose, hemoglobin A1C, and lipid profile.

Depending on what cause is suspected clinically, additional investigations may be performed, including homocysteine levels, testing for thrombotic pathologies (antiphospholipid antibodies, protein S, protein C, antithrombin III, factor V Leiden), tests for rheumatic diseases (eg , determination of antinuclear antibodies, rheumatoid factor, erythrocyte sedimentation rate), serological analysis for syphilis, electrophoresis study of hemoglobin and urine screening test for cocaine and amphetamines.

Thus, for the early diagnosis of diseases of the central nervous system, in particular ischemic stroke, timely and correct collection of anamnesis, including tactile sensations, as well as CT and MRI, and a blood test for prothrombin time/partial thromboplastin time (PT/PTT) is of great importance. fasting blood glucose, hemoglobin A1C and lipid profile.

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