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PATHOGENESIS OF ISCHEMIC STROKE AND MODERN SOLUTIONS FOR CLINICAL DIAGNOSIS

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Abstract: Ischemic stroke is a sudden neurological deficit resulting from focal cerebral ischemia with persistent cerebral infarction (e.g., positive findings on diffusion-weighted MRI). The most common causes are: atherothrombotic occlusion of large arteries; embolism of cerebral vessels (embolic infarction); nonthrombotic occlusion of small, deep cerebral arteries (lacunar infarction); and proximal arterial stenosis with hypotension that reduces cerebral blood flow in arterial bifurcation zones (hemodynamic stroke). In one-third of ischemic strokes, the cause is not identified when the patient is discharged from the hospital; such strokes are classified as cryptogenic. The diagnosis is made on the basis of clinical findings, but CT or MRI is performed to exclude hemorrhage and confirm the infarct size. In some patients, thrombolytic therapy is effective in the acute phase. Depending on the cause of the stroke, carotid endarterectomy or stenting, as well as the use of antiplatelet or anticoagulant drugs, can help reduce the risk of subsequent strokes.

Keywords: Etiology, Pathophysiology, Clinical manifestations, Diagnostics, Treatment, Prognosis Etiology of ischemic stroke

The following modifiable risk factors increase the risk of ischemic stroke:

a) Arterial hypertension
b) Smoking
c) Dyslipidemia
d) Diabetes
e) Insulin resistance
f) Abdominal obesity
g) Obstructive sleep apnea syndrome
h) Alcohol abuse
i) Lack of physical activity
j) High-risk diet (e.g., high in saturated fats, trans fats, and calories)
k) Psychosocial stress (e.g., depression)
l) Heart disease (particularly diseases that predispose to embolism, such as acute myocardial infarction, infective endocarditis, and atrial fibrillation)
m) Carotid artery stenosis
n) Use of certain drugs (e.g., cocaine, amphetamines)

o) Hypercoagulability

p) Vasculitis

q) Use of exogenous estrogen

r) Inevitable risk factors include:

s) History of stroke

t) Floor

u) Race/Ethnicity

v) Old age

w) Family history of stroke

x) Ischemic stroke can be classified as the most common causes of

y) Cryptogenic stroke (i.e., no apparent cardioembolic, lacunar, or atherosclerotic cause; most common classification)

z) Cardioembolism

aa) Lacunar infarctions

bb) Large vessel atherosclerosis (4th place)

Cryptogenic stroke

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

The diagnostic evaluation is incomplete.

Despite an extensive investigation, the cause has not been determined.

There are several possible causes (e.g., atrial fibrillation and ipsilateral carotid artery stenosis).

Embolic stroke of unknown source (ESUS), a subcategory of cryptogenic stroke, is diagnosed when the source is not identified after adequate diagnostic evaluation has excluded lacunar stroke, primary cardioembolic sources, and ipsilateral stenotic-occlusive disease (>50% occlusion). Recent evidence suggests that asymptomatic carotid artery disease with less than 50% occlusion may be an important cause of stroke (1).

Cardioembolism

An embolism can block any part of the arterial system.

Emboli can form as blood clots in the heart chambers, especially when the following factors are present:

Atrial fibrillation

Rheumatic heart disease (usually mitral valve stenosis)

After myocardial infarction

Vegetation on heart valves with bacterial or toxic endocarditis

Atrial myxoma

With prosthetic heart valves

Mechanical circulatory support devices (e.g., left ventricular assist device or LAVA [2])

Other sources of embolism can be blood clots formed during open heart surgery and atheroma of extracranial vessels - the aortic arch and jugular veins. Less common embolisms can be fat (with fractures of tubular bones), gas (with decompression sickness), as well as embolism with venous

thrombi passing from the right half of the heart to the left through the patent foramen ovale (paradoxical embolism). Embolism is interrupted spontaneously or after invasive manipulation of the heart and blood vessels (for example, during catheterization). In rare cases, an embolic stroke develops as a result of thrombosis of the subclavian artery, in which case the vertebral arteries and their branches are subject to embolism.

Lacunar infarctions

Ischemic stroke can also be associated with the formation of lacunar infarcts. These small (≤ 1.5 cm) infarcts are caused by obstruction of the small perforating arteries that supply deep cortical structures. The cause of obstruction of these vessels is believed to be lipoalveolar degeneration (degeneration of the wall of small arteries and their replacement by lipids and collagen). Lacunar infarcts can be caused by embolism. Lacunar infarcts larger than 1.5 cm in patients without cardiovascular risk factors (eg, hypertension, diabetes, smoking) indicate a central source of embolism.

Lacunar infarctions are more common in patients with diabetes mellitus or inadequately controlled hypertension.

Atherosclerosis of large vessels

Large vessel atherosclerosis can affect intracranial or extracranial arteries.

Atherosclerotic plaque, especially ulceration, is a source of thrombus formation. Atherosclerotic plaque formation is possible in any large cerebral artery, 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 occurs in the main trunk of the middle cerebral artery and its branches, as well as in the large arteries of the base of the brain, deep perforating arteries, and small cortical branches. The basilar artery and the supraclinoid part of the internal carotid artery are most often affected, that is, its segment between the cavernous sinus and the supraclinoid process.

Other reasons

Rare causes of stroke include vascular inflammation due to acute or chronic meningitis, vasculitis, and syphilis; dissection of the cerebral arteries or aortic wall; diseases accompanied by hypercoagulability (e.g., antiphospholipid syndrome, hyperhomocysteinemia, presence of a concomitant malignancy); increased blood viscosity (e.g., polycythemia, thrombocytosis, hemoglobinopathy, plasma cell pathology); and rare diseases (e.g., fibromuscular dysplasia, moyamoya disease, Binswanger disease).

Sickle cell disease is a common cause of ischemic stroke in children.

Any factor that compromises the circulatory system (eg, carbon monoxide toxicity, severe anemia or hypoxia, polycythemia, hypotension) increases the risk of all types of ischemic stroke. Strokes can occur in border areas, between the blood flow of individual arteries; blood supply to such areas is usually poor, especially if the patient is hypotensive and/or has narrowed major cerebral vessels.

Most often, ischemic stroke develops due to spasm of blood vessels (for example, after migraine, subarachnoid hemorrhage, use of sympathomimetic drugs or drugs such as cocaine or amphetamines) or thrombosis of venous sinuses (for example, intracranial infection, after surgery, childbirth, secondary hypercoagulability).).

Pathophysiology of ischemic stroke

Insufficient blood flow in a single cerebral artery can often be compensated for 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 cerebral arteries. hemispheres. However, anatomical changes in the diameter of the circle of Willis and collateral vessels, 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 more than 5 minutes, and the size of the lesion depends on the severity of ischemia. With mild ischemia, the process of damage to neural tissue proceeds slowly. Thus, if perfusion is reduced to 40% of normal values, it may take 3-6 hours for all neurons in the ischemic area to die. If severe ischemia lasts > 15-30 minutes, all affected tissue will die (infarct). Damage progresses more rapidly under hyperthermia and more slowly under hypothermia. If tissues are ischemic but still damaged, rapid restoration of blood flow can prevent tissue necrosis or reduce its extent. For example, interventions often have the ability to restore viability to moderately ischemic tissue (the penumbra) surrounded by areas of severe ischemia; the penumbra exists due to collateral blood flow.

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

Many factors can lead to cell death: depletion of adenosine triphosphate (ATP) stores, disruption of ionic homeostasis (including intracellular calcium accumulation), lipid peroxidation by free radicals with damage to cell membranes (an iron-mediated process), exposure to excitatory neurotoxins (e.g. glutamate), and intracellular acidosis due to lactate accumulation.

Signs and symptoms of ischemic stroke

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

Some signs of a stroke

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

In most of these strokes, unilateral neurological symptoms (often starting in one arm and spreading ipsilaterally) develop without headache, fever, or pain in the affected body parts. The development of symptoms is usually gradual, 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 at risk of injury.

Embolic strokes often occur during the day, and the onset of neurological symptoms often precedes the headache. Blood clots usually form at night and are therefore first noticed upon awakening.

Lacunar infarctions may result in one of the classic lacunar syndromes (e.g., pure motor hemiparesis, pure sensory hemianesthesia, combined hemiparesis and hemianesthesia, ataxic hemiparesis, dysarthria, and awkward hand syndrome); there are no signs of cortical dysfunction (e.g., aphasia). The consequence of recurrent lacunar infarctions may be the development of postinfarction dementia.

When a stroke occurs, an embolic stroke is more likely to occur than a thrombotic stroke. Seizures may occur months or years later; late attacks result from scarring or hemosiderin deposition at the site of ischemia.

Increasing neurological deficit, especially impaired consciousness in the first 48-72 hours, is often associated with increasing brain edema, but may also be associated with an expansion of the infarct zone. If the infarct is small, functional improvement is noticeable in the first days of illness; further recovery occurs gradually up to 1 year.

Diagnosis of ischemic stroke

Initial clinical examination

Neuroimaging and blood glucose level determination using test strips

Test to determine the cause of a stroke

The diagnosis of ischemic stroke should be suspected when there is a sudden onset of neurological symptoms that are localized to the area of blood supply to one of the cerebral arteries. Ischemic stroke should be distinguished from other causes of similar focal deficits (sometimes called stroke mimics, which are non-cerebral vascular disorders that cause focal neurological signs (e.g., hypoglycemia), e.g.

seizures (e.g., with postictal paralysis)

Infectious lesion of the central nervous system

Functional neurological disorders (usually diagnosed by exclusion)

Migraine (e.g., 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 can be particularly useful as an outcome measure in efficacy studies. The National Institutes of Health Stroke Scale (NIHSS) is a 15-point scale that assesses a patient's level of consciousness, speech function, and motor and sensory impairment by asking the patient to answer questions and perform physical and mental tasks. It is also useful for selecting appropriate treatment and predicting outcome.

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

It is not clear how to differentiate the different types of stroke based on clinical presentation; however, some guidelines based on the progression of symptoms, time of onset, and type of impairment can aid in diagnosis.

Although the initial diagnosis is made by clinical signs, neuroimaging and blood glucose level determination using test strips are considered urgent measures.

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

Heart (e.g., atrial fibrillation, potential systemic features for embolism)

Vascular disease (e.g., critical arterial stenosis identified on vascular imaging)

Blood diseases (e.g., diabetes, dyslipidemia, hypercoagulation)

With cryptogenic strokes, the cause cannot be determined.

Brain assessment

Neuroimaging with CT or MRI is primarily performed to exclude intracerebral hemorrhage, subdural or epidural hematoma, and rapidly growing, bleeding, or suddenly symptomatic tumors. CT findings may be unremarkable in the first few hours, even in the case of a large ischemic stroke in the carotid artery. Changes may include loss of sulcus or insular cortex, loss of gray-white matter junction between cortex and white matter, and increased density of the middle cerebral artery. Medium- to large-sized infarcts are visible as areas of decreased density within 6 to 12 hours after ischemia. Small infarcts (eg, lacunar) can only be detected with MRI.

Diffusion-based MRI (a very sensitive test for early ischemia) can be performed immediately after the initial CT scan.

This CT scan shows a well-defined low-density lacunar infarction (arrow) within the basal ganglia.

Pathology of the cardiovascular system

Cardiac evaluation typically includes ECG, telemetry or Holter monitoring, serum troponin levels, and transthoracic or transesophageal echocardiography. Implantable cardiac monitors are useful in identifying underlying atrial arrhythmias in patients with cryptogenic stroke (1).

Vascular diseases

Vascular imaging may include magnetic resonance angiography (MRA), computed tomography angiography (CTA), bilateral carotid and transcranial duplex ultrasound, and conventional angiography. The choice and sequencing of imaging tests are individualized based on clinical findings. MRA, CT-A, and carotid ultrasound provide equally good visualization of the anterior cerebral circulation (carotid basin); however, MRA and CT-A provide better visualization of the posterior cerebral circulation than carotid ultrasound. In general, CTA is preferred over MRA because it avoids motion artifacts. In general, CTA or MRA should be performed promptly, but treatment with IV tissue plasminogen activator (tPA) should not be delayed if indicated.

Effects of thrombectomy after stroke (arteriogram)

Blood-related causes

Blood tests are performed to identify causes related to blood disorders (such as thrombotic pathologies), to assess the contribution of these pathologies and the contribution of other causes. Routine testing usually includes a complete blood count (CBC), blood chemistry panel, prothrombin time/partial thromboplastin time (PT/PTT), fasting glucose, hemoglobin A1C, and lipid profile.

Depending on the clinically suspected cause, additional tests may be performed, including homocysteine levels, thrombotic pathologies (antiphospholipid antibodies, protein S, protein C, antithrombin III, factor V Leiden), tests for rheumatic diseases (e.g., antinuclear antibodies, rheumatoid factor, erythrocyte sedimentation rate), serological analysis for syphilis, hemoglobin electrophoresis, and urine screening tests for cocaine and amphetamine.

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