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TREATMENT OF CEREBRAL INFARCTION AT THE PREHOSPITAL STAGE AND IN A SPECIALIZED DEPARTMENT

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Abstract:

During hospitalization, a complex of emergency medical measures is carried out, regardless of the nature of the brain infarction (basic and differentiated therapy). Basic undifferentiated therapy provides a set of emergency treatment measures aimed at stabilizing vital functions, regardless of the nature of a brain infarction. The complex includes measures providing for monitoring the function of the respiratory and cardiovascular systems and their correction, regulation of blood pressure, correction of glucose metabolism, maintenance of water-electrolyte balance, maintenance of normal body temperature, treatment of dysphagia, provision of appropriate nutrition.

Keywords: *brain infarction, prehospital therapy, first aid.*

Introduction

Currently, brain infarction remains a significant medical and socio-economic problem. At least 15 million brain infarctions are registered annually in the world, and a brain infarction occurs for 40 seconds, ranking 2nd after dementia in the list of the main causes of disability. Given these prerequisites, the issues of effective treatment of this formidable disease are extremely relevant.

The main directions of therapy for cerebral infarction follow from the recommendations of the European Stroke Organization (ESO) Executive Committee; ESO Writing Committee, 2008), the National Association for the Fight against Cerebral Infarction (NABI), the Scientific Center of Neurology.

A cerebral infarction is an acute violation of cerebral circulation, characterized by sudden (within minutes, less often – hours) the appearance of focal neurological symptoms (motor, speech, sensory, coordination, visual and other disorders) and/or general cerebral disorders (changes in

consciousness, headache, vomiting, etc.) that persist for more than 24 hours or lead to the death of the patient in a short period of time due to the cause of cerebrovascular origin.

The clinical and pathogenetic forms of cerebral infarction are distinguished:

1) ischemic cerebral infarction (cerebral infarction) caused by acute focal cerebral ischemia leading to a heart attack (zone of ischemic necrosis) of the brain. The heterogeneity of ischemic cerebral infarction is represented by pathogenetic subtypes (Vereshchagin N.V. et al., 2000): 1) atherothrombotic (34% of cases); 2) cardioembolic (22%); 3) hemodynamic (15%); 4) lacunar (22%); 5) cerebral infarction by type of hemorheological microocclusion (7%).

Hemorrhagic brain infarction (non-traumatic intracerebral hemorrhage) caused by rupture of an intracerebral vessel and penetration of blood into the parenchyma of the brain or rupture of an arterial aneurysm with subarachnoid hemorrhage.

3) venous cerebral infarction develops more often in patients at a relatively young age with a premorbid burden – constitutional venous insufficiency and cerebral venous dyscirculation. It is characterized by subacute, slow development with a predominance of cerebral symptoms over focal in the clinical picture, followed by a relatively rapid regression of cerebral symptoms. Focal neurological deficit, in turn, is caused by the localization of focal brain damage, which, as a rule, does not coincide with the "pools" of blood supply to the main intracranial arteries. In this case, the parietal-occipital region with a bilateral location of venous infarcts is most often affected, which is due to the anatomical features of cerebral venous outflow. On MRT, one can see heterogeneous irregularly shaped foci with uneven, indistinct contours, signs of cerebral venous dyscirculation and intracranial venous stagnation, as well as signs of vasogenic edema appearing already on the first day of the disease.

acute cerebrovascular accident also includes transient disorders of cerebral circulation, characterized by the sudden appearance of focal neurological symptoms in a patient with cardiovascular disease (hypertension, atherosclerosis, atrial fibrillation, vasculitis, etc.), lasting several minutes, less often hours, but no more than 24 hours, followed by complete restoration of impaired functions.

Transient disorders of cerebral circulation include:

1) transient ischemic attack (TIA), which develops as a result of short-term local cerebral ischemia and is characterized by sudden transient neurological disorders with focal symptoms;

2) hypertensive cerebral crisis, which is a condition associated with an acute, usually significant rise in blood pressure (BP) and accompanied by the appearance of cerebral (less often focal) neurological symptoms secondary to hypertension. The most severe form of hypertensive crisis is acute hypertensive encephalopathy, the basis of the pathogenesis of which is cerebral edema.

If the episode assessed as a new acute cerebrovascular accident occurred within 28 days of the onset of the registered cerebral infarction, it is considered as a continuation of the primary attack. A brain infarction that occurred after 28 days from the beginning of the first brain infarction in this patient is considered as a repeated brain infarction.

The main task at the prehospital stage is the correct and rapid diagnosis of acute cerebrovascular accident, which is possible on the basis of clarifying complaints, anamnesis and conducting a somatic and neurological examination. An accurate determination of the nature of a cerebral infarction (hemorrhagic or ischemic) is not required, it is possible only in a hospital after CT or MRT examinations of the brain.

A brain infarction should be suspected in all cases in the presence of acute development of focal neurological symptoms (within minutes, less often hours) or a sudden change in the level of conani.

Acute development of cerebral or meningeal symptoms is possible.

The optimal time for hospitalization of patients with acute cerebrovascular accident is the first 3-6 hours in a specialized department for the treatment of acute cerebral circulatory disorders.

It is recommended to notify the receiving party in advance, indicating the approximate time of the patient's arrival. With later hospitalization, the number of complications of cerebral infarction and the severity of subsequent disability of patients with acute cerebrovascular accident significantly increases.

A relative contraindication to hospitalization is terminal coma, a history of dementia, and the terminal stage of cancer. Patients with acute cerebrovascular accident who have remained on outpatient treatment for various reasons should be prescribed basic, symptomatic and neuroprotective therapy during the first day.

TREATMENT OF acute cerebrovascular accident AT THE PREHOSPITAL STAGE

Correction of blood pressure. Aggressive reduction of blood pressure in cerebral infarction is unacceptable due to the risk of a drop in cerebral perfusion, therefore, the use of drugs leading to a sharp decrease in blood pressure is contraindicated: nifedipine, ganglioblockers, active peripheral vasodilators. A gradual decrease in blood pressure is shown only at figures exceeding 200/110 mmHg. It is necessary to limit intravenous bolus and sublingual administration of antihypertensive drugs. In this case, preference should be given to antihypertensive drugs from the ACE inhibitor group: captopril, enalapril, etc.

If hospitalization is delayed by more than 30 minutes, infusion therapy should be activated. The main infusion solution should be considered 0.9% sodium chloride solution.

In the case of generalized convulsive seizures, diazepam 10 mg I / v is used slowly for their relief, if ineffective, repeatedly (10 mg I / v) after 3-4 minutes. It must be remembered that the maximum daily dose of diazepam is 80 mg.

The most common errors of therapy are cerebral infarction at the prehospital stage:

1. The use of hemostatic therapy (calcium chloride, aminocaproic acid or ascorbic acid) during tracheal intubation. It is important to maintain adequate ventilation, especially during sleep, when episodes of apnea or hypopnea are possible.

2. Correction of the function of cardio-dopamine and sufficient fluid (0.9% sodium chloride solution) in general treatment.

3. Correction of water-electrolyte metabolism disorders. Violations are suspected of hemorrhagic cerebral infarction, because these drugs begin to act after a few days, and their use in acute cerebrovascular accident has not been studied or is contraindicated.

2. The use of antihypertensive drugs that sharply reduce blood pressure, which, in conditions of disruption of autoregulation of cerebral circulation, creates a real threat of hypoperfusion of the brain and expansion of the volume of its damage.

3. The appointment of acetylsalicylic acid and other antiplatelet agents due to the impossibility of reliably excluding cerebral hemorrhage.

4. The use of furosemide for the treatment of cerebral edema due to a possible sharp decrease in blood pressure and exacerbation of cerebral ischemia, as well as due to the development of hemoconcentration.

5. The use of a number of nootropics (piracetam, instenone, nicotinol, picamilon, etc.), which leads to excessive stimulation and exhaustion of the brain under conditions of anaerobic glycolysis.

TREATMENT OF CEREBRAL INFARCTION IN A SPECIALIZED DEPARTMENT, a complex of emergency medical measures is carried out During hospitalization, regardless of the nature of the cerebral infarction (basic and differentiated therapy). Basic undifferentiated

The therapy provides a set of emergency treatment measures aimed at stabilizing vital functions, regardless of the nature of a brain infarction. The complex includes measures providing for monitoring the function of the respiratory and cardiovascular systems and their correction, regulation of blood pressure, correction of glucose metabolism, maintenance of water-electrolyte balance, maintenance of normal body temperature, treatment of dysphagia, provision of appropriate nutrition.

1. Correction of respiratory disorders. It is necessary to control the level of blood oxygenation. In cases of hypoxemia: coma (≤8 points on the Glasgow Coma Scale), bradypnea (<12 in 1 minute), tachypnea (>35-40 in 1 minute), loss of stem reflexes, endotracheal intubation is performed. It is important to maintain adequate ventilation, especially during sleep, when episodes of apnea or hypopnea are possible.

2. Correction of the cardiovascular system function. The greatest hemodynamic disorders occur during 2-7 days of the disease, therefore, especially in the first days of the development of a cerebral infarction, constant monitoring of the ECG and pulse is necessary. In case of cardiac arrhythmia, antiarrhythmic drugs are prescribed. Blood pressure reduction, according to existing recommendations, should be carried out only if, upon repeated measurement, the blood pressure exceeds 220 mmHg, or diastolic –120-140 mmHg, or average more than 130 mmHg. At the same time, the decrease in blood pressure should not exceed 10-15% of the initial level. However, an increased blood pressure level has a protective effect on ischemic brain tissue only in the acute period of cerebral infarction, and after 2-3 days. or 1 week after a brain infarction begins to have a damaging effect (increases vasogenic edema, BBB permeability), therefore, planned hypotensive therapy should be performed. Recommended medications for this are: perindopril 5 mg daily, captopril 25-50 mg, indapamide 1.5 mg. Urgent antihypertensive therapy is required for heart failure, aortic dissection, acute myocardial infarction, acute renal failure, the need for thrombolysis or intravenous heparin treatment. In case of arterial hypotension, the use of dopamine and a sufficient amount of liquid (0.9% sodium chloride solution) in general treatment is indicated.

3. Correction of violations of water-electrolyte metabolism. Violations of the water-electrolyte balance occur in 85% of patients with acute cerebral infarction. In the most severe patients, water deficiency is noted on day 2-5 with loss of 2-6% of fluid due to insufficient intake into the body, as well as loss of fluid through the skin and respiratory tract with fever, shortness of breath, ventilation, breathing through a tracheostomy. Daily administration of 2000-2500 ml of liquid parenterally throughout the day in 2-3 doses is indicated. Excessive positive water balance should be avoided, as this can lead to pulmonary edema and increased cerebral edema. Therefore, in patients with cerebral edema, it is necessary to maintain 300-350 ml of negative fluid balance (minus 300-350 ml of fluid per day).

4. Treatment of dysphagia and provision of appropriate nutrition. The prevalence of dysphagia in patients with cerebral infarction reaches 47% and is both a risk factor for aspiration and dehydration, and the cause of impaired normal fluid and food intake with the development of exhaustion and dehydration. At the same time, adequate nutritional support can compensate for increased energy consumption in case of a brain infarction and provide the body in critical condition with the necessary plastic substrates. In this regard, all patients with cerebral infarction should evaluate the function of swallowing and, if dysphagia is detected, take this symptom into account when feeding. In cases of severe dysphagia, enteral nutrition is recommended, and if this is not possible, they switch to probe nutrition.

Differentiated treatment of hemorrhagic stroke. In hemorrhagic cerebral infarction, hypotensive therapy is carried out more actively than in ischemic. However, even in this case, it is carried out carefully, as it can significantly worsen cerebral hemocirculation, especially in conditions of developed intracranial hypertension. In such cases, a moderate decrease in blood pressure is combined with dehydration therapy.

At this stage of the disease, droperidol is prescribed 2 ml of 0.25% solution intravenously. In case of a significant increase in blood pressure, pentamine is used – 1 ml of 5% solution in 250 ml of isotonic sodium chloride solution intravenously, furosemide (lasix) – 2-4 ml of 1% solution intravenously or intramuscularly. Basic therapy measures aimed at reducing intracranial pressure continue to be carried out:

mannitol 1.0–1.5 g / kg per day in the form of a 15-20% solution, furosemide 40 mg intravenously drip for 5 days.

The appointment of steroids in this case is not shown.

Patients with hemorrhagic cerebral infarction are shown the appointment of neuroprotective drugs, as well as antioxidants.

The most effective are ceraxone (citicoline) – 1000-2000 mg intravenously, and actovegin – 400-800 mg (10-20 ml) intravenously in 200 ml of isotonic sodium chloride solution for 10-14 days.

Treatment for subarachnoid hemorrhage is carried out according to the same scheme as for cerebral hemorrhage. In addition, with intense headache intravenously, baralgin is administered - 5 ml or 4 ml of 50% analgin solution with 1-2 ml of 1% diphenhydramine solution; with indomitable vomiting, haloperidol 1-2 ml of 0.5% solution or droperidol 1-2 ml of 0.25% solution is prescribed intramuscularly; if seizures or psychomotor agitation occur, diazepam 2-4 ml of 0.5% solution is intravenously administered. It is important to observe absolute rest for 6-8 weeks.

In order to prevent or relieve cerebral angiospasm after subarachnoid hemorrhage and prevent the development of delayed after a cerebral infarction in the pool of the affected artery, a calcium antagonist with a brain effect of nimotop (nimodipine) is prescribed – 60 mg every 4-6 hours for 10-14 days. Only in case of repeated hemorrhage, aminocaproic acid (30 g per day) is administered for at least 3 weeks within 2 weeks.

Differentiated treatment of ischemic stroke includes two main approaches:

a) restoration of blood flow in an ischemic area of the brain or recanalization of an infarct-dependent cerebral artery by thrombolysis;

c) neuroprotection (primary and secondary) with elements of secondary prevention.

Recanalization of infarct-dependent cerebral artery. Intravenous administration of rt-PA for recanalization was the first drug treatment for acute ischemic cerebral infarction with a prescription of up to 4.5 hours, which was found effective in randomized clinical trials.

The recombinant tissue plasminogen activator rt-PA (actilize) follows

apply within a 4.5-hour "therapeutic window" with a dose of 0.9 mg / kg, the maximum dose is 90 mg, of which 10% is administered intravenously bolus for 1-2 minutes. with the next intravenous infusion for 60 minutes.

The main contraindications to thrombolysis are:

the presence of intracranial hemorrhage according to CT or MRI data; minimal neurological deficit regressing before the start of treatment; severe brain infarction with a neurological deficit level on the NIHSS scale of 25 points or more, i.e. in the case of a large-focal heart attack, verified according to CT or MRI data; epileptic seizure at the beginning of development of a brain

infarction; a previous heart attack brain, as well as in the presence of concomitant diabetes mellitus; previously suffered a brain infarction over the past 3 months; a brain infarction that occurred after waking up; systolic blood pressure greater than 185 mmHg or diastolic blood pressure greater than 110 mmHg, blood glucose level less than 3 or more than 22 mmol/l; high risk of hemorrhagic complications; severe concomitant diseases; heparin treatment during the previous 48 hours.

A dangerous complication of thrombolytic therapy in the case of acute cerebral infarction is the development of fatal intracerebral hemorrhage or symptomatic hemorrhagic transformation of a cerebral infarction. Asymptomatic hemorrhagic transformation is considered a marker of reperfusion, and it can be associated with favorable clinical consequences.

In acute ischemic cerebral infarction, early use of heparin and heparinoids in therapeutic doses is not recommended (ESO, 2008). The generally accepted indications for the use of preventive doses of heparin after the development of acute ischemic cerebral infarction are the following: high risk of deep vein thrombosis of the lower extremities or pulmonary embolism; cardioembolic ischemic stroke with a high risk of reembolization, retrombosis (atrial fibrillation, artificial valves) after exclusion of hemorrhagic transformation; it is preferable to start with warfarin immediately than switch from heparin to warfarin (ESO, 2008); acquired or hereditary coagulations (deficiency of proteins C and S, antiphospholipid syndrome); symptomatic extra- or intracranial stenosing processes (stenosis of the internal carotid artery, repeated TIA or progressive cerebral infarction);

symptomatic dissection of the extracranial arteries. Preventive heparin therapy is performed only 24 hours after thrombolysis.

In venous cerebral infarction, anticoagulant therapy is the main one and lasts at least 9-12 months. The international Normalized Ratio (MHO) prothrombin test is used as a control. The target MHO level is 2.0–3.0. In acute ischemic cerebral infarction of non-cardiac etiology, platelet inhibitors should be included, not heparin. Of the drugs in this group, acetylsalicylic acid (aspirin) is the most studied.

The drug is prescribed at a dose of 160-325 mg / day. during the first 48 hours immediately after the onset of the first symptoms of a cerebral infarction; it is not used if thrombolytic therapy is planned; in this case, aspirin is prescribed only 24 hours after its implementation (ESO, 2008). Patients who do not tolerate aspirin should receive alternative antiplatelet agents (clopidogrel 75 mg1 once a day). Neuroprotective therapy. Among the existing medicines with

The largest number of randomized placebo-controlled clinical trials (more than 10) have passed ceraxone (citicoline) for the alleged neuroprotective effect. The drug has a high level of evidence-based neuroprotective activity in the treatment of acute ischemic cerebral infarction, with the achievement of maximum therapeutic effect when prescribed in the first 24 hours. Magnesium sulfate is often used intravenously for 10-20 ml of primary neuroprotection as a non-competitive antagonist of NMDA receptors. At the same time, a multicenter trial based on intravenous administration of magnesium sulfate in the first 12 hours after the development of a completed ischemic cerebral infarction followed by a 24-hour infusion of the drug did not have a positive clinical effect, except in patients with lacunar infarction.

Secondary neuroprotection of ischemic cerebral infarction is performed with the use of antioxidants, the mechanism of which is based on inhibition of reactions of non-enzymatic free radical oxidation of lipids and biopolymers, proteins, mucopolysaccharides and nucleic acids, followed by a membrane-protective effect, which ensures stabilization of structural integrity and preservation of functional properties of neurons.

Antioxidants with neuroprotective properties include: cavinton, cytoflavin, actovegin, mildronate, tocopherol acetate (vitamin E). Drugs with pronounced cholinergic and neurotrophic effects (choline alfoscerate), as well as neuropeptides (cortexin, cerebrolysin) are also used.

Treatment in case of complications of cerebral infarction of the brain. Among the complications of a cerebral infarction, cerebral edema, deep vein thrombosis, pulmonary embolism, pneumonia, epileptic seizures, urinary tract infections, bedsores are most often observed. Cerebral edema mainly occurs during the first 24-72 hours after the development of ischemic cerebral infarction.

Drug treatment provides treatment with saluretics (furosemide 40 mg 2-3 times a day) with vasogenic and osmotherapy (mannitol 25-50 g every 3-6 hours) for interstitial edema of the brain. With the development of post-ischemic cerebral edema, hypotonic, glucose-containing solutions, as well as corticosteroids, are not recommended.

Unfortunately, to date, cerebral infarction remains the most serious disease, entailing enormous losses for society. At the same time, the number of people of working age is constantly growing among the sick. The main task of a doctor in this situation is to accurately diagnose, determine the optimal strategy and tactics for treatment and further prevention of the disease. At the same time, an early start and a sufficient amount of treatment carried out in specialized institutions focused on modern evidence-based medicine is key in reducing mortality in cerebral infarction, allows you to restore lost functions and return patients to a full life in the vast majority of cases.

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