American Journal of Medicine and Medical Sciences 2025, 15(3): 642-646
DOI: 10.5923/j.ajmms.20251503.33

Treatment of Patients with Cerebral Infarction at the Prehospital Stage and in a Specialized Department

Urinov Muso Boltaevich*, Akhtamov Tursunpulat Umarovich

Bukhara State Medical Institute named after Abu Ali Ibn Sino, Bukhara, Uzbekistan

Abstract Stroke is currently a serious medical and socio-economic problem. At least 15 million strokes are registered worldwide every year. In Russia, one stroke occurs every 40 seconds, making it the second leading cause of disability after dementia. The cost of treatment for a stroke patient in Russia is about 127,000 rubles per year, including inpatient treatment, medical and social rehabilitation and secondary prevention. Given these prerequisites, the issue of effective treatment of this terrible disease is extremely important.

Keywords Cerebral infarction, Prehospital stage

The main directions of stroke treatment are based on the recommendations of the Executive Committee of the European Stroke Organization (ESO) (ESO Writing Committee, 2008), the National Stroke Association (NSA) and the Center for Neurology. Stroke is an acute cerebrovascular accident (CSC) that occurs suddenly (within a few minutes, rarely for hours). The appearance of focal neurological symptoms (motor, speech, sensory, coordination, visual and other disorders) and/or generalized cerebral disorders (changes in consciousness, headache, vomiting, etc.) lasting more than 24 hours, or caused by cerebral is characterized by death within a short period of time due to vascular causes [2,5].

The following clinical types and pathologies of stroke are distinguished: 1) ischemic stroke caused by acute focal cerebral ischemia (cerebral infarction); 2) cerebral infarction (ischemic-necrotic areas). The diversity of ischemic stroke is expressed in pathological subtypes: 1) atherothrombotic 34% of cases, 2) cardioembolic 22%, 3) hemodynamic 15, 4) lacunar 22%, 5) stroke due to hemodynamic microclusion 7%; 2) hemorrhagic stroke due to rupture of an intracerebral vessel and penetration of blood into the parenchyma of the brain or rupture of an aneurysm with subarachnoid hemorrhage (non-traumatic intracerebral hemorrhage); 3) venous stroke, which occurs more often in relatively young patients with premorbid disorders such as constitutional venous insufficiency or disorders of cerebral venous circulation. It is characterized by a subacute, slow onset, the predominance of cerebral symptoms over focal ones in the clinical picture, and then a relatively rapid regression of cerebral symptoms. Focal neuropathy is caused by the localization of focal brain lesions, which, as a rule, do not coincide with the "pool" of blood supply to the main intracranial arteries [3]. Bilateral venous infarctions are most common in the parietal and occipital regions, which is due to the anatomical features of the venous outflow of the brain.

On MRT, one can see heterogeneous foci of irregular shape with uneven and indistinct contours, signs of impaired cerebral venous circulation and intracranial venous stasis, as well as signs of angiogenic edema, which may appear on the first day after the onset of the disease [1,7,10]. Transient disorders of cerebral circulation, characterized by the sudden appearance of focal neurological symptoms in patients with cardiovascular diseases (hypertension, arteriosclerosis, atrial fibrillation, vasculitis, etc.), lasting several minutes, less often several hours, but lasting more than 24 hours, followed by a complete restoration of impaired functions, are also subject to CMR. Transient disorders of cerebral circulation include: 1) transient ischemic attacks (TIA), which develop as a result of short-term focal cerebral ischemia and are characterized by a sudden transient neurological deficit with focal symptoms; 2) hypertensive cerebral crisis is a condition accompanied by an acute, usually pronounced increase in blood pressure (BP), which with the appearance of generalized cerebral (often focal) neurological symptoms, secondary to hypertension. The most severe form of hypertensive crisis is acute hypertensive encephalopathy, the etiology of which is based on cerebral edema [1,4,12].

If a new episode with a STEMI score occurred before the 28th day after the onset of the stroke, it is considered a continuation of the primary stroke. Strokes that occur after 28 days from the onset of the first stroke in this patient are considered recurrent strokes. The main task at the prehospital stage is the accurate and rapid diagnosis of STEMI, which can be based on clarifying complaints, medical history, physical and neurological examination. There is no need to determine the exact nature of the stroke (hemorrhagic or

* Corresponding author:
urinov.muso@bsmi.uz (Urinov Muso Boltaevich)
Received: Feb. 1, 2025; Accepted: Feb. 26, 2025; Published: Mar. 10, 2025
Published online at http://journal.sapub.org/ajmms

ischemic), which is possible only after CT or MRI of the brain in a hospital. In any case, a stroke should be suspected when focal neurological symptoms appear (within a few minutes, less often hours) or a sudden change in the level of consciousness. Cerebral and meningeal symptoms may also become acute [3,7,13].

The optimal time for admission of patients with acute cerebral hemorrhage is the first 3-6 hours to the department specializing in the treatment of acute cerebral circulatory disorders. It is recommended to inform the receiving department in advance about the approximate time of admission of the patient. Late hospitalization significantly increases the number of stroke complications and the severity of subsequent disability in patients with STEMI. Relative contraindications to hospitalization are terminal coma, dementia with amnesia, and terminal tumor diseases.

Patients with STEMI who remain on outpatient treatment for various reasons should receive basic symptomatic and neuroprotective therapy during the first 24 hours. Due to the risk of decreased cerebral perfusion, aggressive lowering of blood pressure in stroke is unacceptable, and the use of drugs that cause a rapid decrease in blood pressure (nifedipine, ganglioblockers, active peripheral vasodilators) is contraindicated. Blood pressure should be lowered gradually, only when it exceeds 200/110 mmHg. Intravenous bolus or sublingual administration of drugs that lower blood pressure should be limited. In this case, preference should be given to antihypertensive drugs from the ACE inhibitor group, such as captopril and enalapril. If admission is delayed by more than 30 minutes, infusion therapy should be performed. The main infusion solution should be 0.9% sodium chloride solution. In case of generalized seizures, diazepam 10 mg should be slowly administered intravenously, and if ineffective, repeat the administration of 10 mg intravenously after 3-4 minutes. Remember that the maximum daily dose of diazepam is 80 mg [4,9].

The most common mistakes in the treatment of stroke at the prehospital stage: 1. The use of hemostatic therapy (calcium chloride, aminocaproic acid, ascorbic acid) with vitamin B complexes with a neurostimulating effect. These drugs begin to act after a few days, presumably cause hemorrhagic stroke, and their use in STEMI has not been studied or contraindicated; 2. The use of antihypertensive drugs that rapidly reduce blood pressure. With impaired autoregulation of cerebral circulation, there is a real threat of hypoperfusion of the brain and an increase in the volume of its damage. 3. acetylsalicylic acid or other anticoagulants, as brain hemorrhage cannot be ruled out with certainty. 4. The use of furosemide for the treatment of cerebral edema, in which blood pressure decreases rapidly and cerebral ischemia worsens. 5, and possible hemoconcentration; 5. The use of many psychotropic drugs (for example, piracetam, instenone, nicotinol, picamilon) leads to overexcitation and exhaustion of the brain under conditions of anaerobic glycolysis. Stroke treatment in specialized departments, a number of urgent medical measures are carried out during hospitalization, regardless of the nature of the stroke (basic and differentiated treatment). Basic undifferentiated treatment provides for a number of urgent therapeutic measures aimed at stabilizing vital functions, regardless of the nature of the stroke. These include monitoring and correcting respiratory and cardiovascular function, regulating blood pressure, correcting glucose metabolism, maintaining water and electrolyte balance, maintaining normal body temperature, treating dysphagia, and ensuring adequate nutrition. 1. Correction of respiratory disorders. It is necessary to monitor the level of blood oxygenation. In case of hypoanemia: endotracheal intubation in the presence of coma (< 8 points on the Glasgow coma scale), bradycardia (<12 in 1 minute), tachypnea (>35-40 beats per minute) or loss of stem reflexes. It is important to maintain adequate ventilation, especially during sleep, as episodes of apnea and hypopnea may occur; 2. Correction of the function of the cardiovascular system. Since hemodynamic disorders are most likely between 2 and 7 days after the onset of stroke, constant monitoring of the ECG and pulse is necessary, especially in the early stages of stroke development. In case of cardiac arrhythmia, antiarrhythmic drugs are prescribed. According to current recommendations, blood pressure reduction should be carried out only if repeated SBP readings exceed 220 mmHg. or diastolic - 120-140 mmHg with an average of 130 mmHg. At the same time, the decrease in blood pressure should not exceed 10-15% of the initial value. However, routine antihypertensive therapy is necessary because hypertension protects ischemic brain tissue only in the acute phase of stroke, and the damaging effects (increased vasogenic edema, increased BBB permeability) begin 2-3 days or a week after the stroke. Recommended medications for these purposes are perindopril 5 mg / day, captopril 25-50 mg and indapamide 1.5 mg. Urgent antihypertensive therapy is necessary for heart failure, aortic dissection, acute myocardial infarction, acute renal failure, thrombolytic therapy or intravenous heparinization. In case of arterial hypotension, the indications for systemic treatment are dopamine and adequate infusion (0.9% sodium chloride solution); 3. Correction of disorders of water and electrolyte metabolism. Water-electrolyte imbalance occurs in 85% of patients in the acute phase of stroke. On day 2-5, the most severely affected patients experience water deficiency with a loss of 2-6% of water due to insufficient fluid intake into the body, as well as water loss through the skin and respiratory tract with fever, shortness of breath, artificial respiration and tracheostomy breathing of 2000-2500 ml of liquid per day in two or three divided parenteral doses. Percentage of fluid loss. Excessive water withdrawal should be avoided, as this can cause pulmonary edema and increase brain edema. Therefore, in patients with cerebral edema, a negative fluid balance should be maintained at the level of 300-350 ml (300-350 ml of fluid minus per day) [6,10,13].

The introduction of solutions containing glucose is impractical due to the risk of hyperglycemia. It should be remembered that infusion therapy should be performed while monitoring electrolytes, acid-base state, plasma protein content, glucose and other biochemical parameters. 4. Regulation of glucose metabolism Hyperglycemia (blood glucose >7 mmol/L) in

the acute phase of stroke has an adverse effect on neuron cells in the ischemic semi-infarction zone. This worsens the prognosis, as it increases the area of the infarction. In this case, treatment with insulin is necessary. Along with this, a drop in blood glucose levels also has a very negative effect on the condition of brain cells. To correct hypoglycemia, 10-20% glucose should be administered intravenously by bolus or intravenous infusion, and glucose solution and 100 g of thiamine together should be administered to patients with cachexia or alcoholism. 5. Maintaining a normal body temperature and suppressing fever. It is important to reduce body temperature in a timely manner, as an increase in body temperature above 37.5℃ within 24 hours after the onset of the disease exacerbates brain damage. Antibiotic therapy is prescribed only to patients with a confirmed infection. Antiviral drugs are not indicated. 6. Treatment of dysphagia and adequate nutrition. The prevalence of dysphagia in stroke patients reaches 47% and is a risk factor for aspiration and dehydration, as well as the cause of impaired normal intake of fluids and food with the onset of exhaustion and dehydration. At the same time, adequate nutrition can compensate for increased energy consumption during stroke and provide the body in critical condition with the necessary plastic substrates. In this regard, all patients who have suffered a stroke should evaluate the function of swallowing and, if dysphagia is detected, the diet should take this symptom into account. In case of severe dysphagia, enteral nutrition is recommended, and if this is not possible, the patient should be transferred to nutrition through a probe.

Differential treatment of hemorrhagic stroke In hemorrhagic stroke, antihypertensive therapy is more aggressive than in ischemic stroke. However, even in this case, it is carried out with caution, especially in the presence of progressive intracranial hypertension, as it can significantly impair cerebral circulation. In such cases, moderate reduction of blood pressure and dehydration therapy should be combined. At this stage, 2 ml of 0.25% droperidol solution should be administered intravenously. With a marked increase in blood pressure, 1 ml of pentamine, a 5% solution dissolved in 250 ml of isotonic sodium chloride solution, and 2-4 ml of furosemide (lasix), a -1% solution, are administered intravenously or intramuscularly. Continue the basic therapy aimed at reducing intracranial pressure: mannitol 1.0-1.5 g / kg once a day in the form of a 15-20% solution and furosemide 40 mg intravenously for 5 days. Steroids are not prescribed in this case. Neuroprotective and antioxidant drugs are indicated for patients with hemorrhagic stroke. The most effective are seraxone (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. To prevent or relieve cerebral vascular spasm after subarachnoid hemorrhage and prevent the development of delayed cerebral infarction in the affected artery basin, nimotop (nimodipine)-60 mg, a calcium channel blocker acting in the brain, every 4-6 hours for 10-14 days Only if the hemorrhage recurs within 2 weeks, Aminocaproic acid (30 g per day) for at least 3 weeks. Surgical treatment of parenchymal hemorrhage is performed in the presence of a lateral hematoma (40 ml) and hemorrhage into the cerebellum. In subarachnoid hemorrhage, priority is given to endovascular treatment aimed at detaching the ruptured aneurysm from the arterial bed: embolization, balloon implantation, stenting, and spiral insertion [5,8,12,14].

Contraindications to surgery are coma, impaired vital functions, and blood flow to the ventricles (severity III and IV on the Gantt and Hess scales). There are two main approaches to the differentiated treatment of ischemic stroke: a) restoration of blood flow in ischemic areas of the brain or recanalization of infarct-dependent cerebral arteries using thrombolytic therapy; c) neuroprotection (primary and secondary), including elements of secondary prevention. Recanalization of infarct-dependent cerebral arteries. Intravenous administration of rt-PA for recanalization is the first pharmacological treatment for acute ischemic stroke for 4.5 hours and was confirmed in a randomized clinical trial. Recombinant tissue plasminogen activator rt-PA (Actilise) is administered at a dose of 0.9 mg / kg, a maximum of 90 mg for 4.5 hours, 10% of which is administered intravenously by bolus infusion for 1-2 minutes, followed by intravenous infusion for 60 minutes. The main contraindications to thrombolytic therapy are the presence of intracranial hemorrhage according to CT or MRI, recovery of minimal neurological disorders before treatment, severe stroke with neurological disorders ≥25 points on the NIHSS scale, i.e. extensive regional stroke In the case of extensive regional stroke confirmed by CT or MRI, cerebral epileptic seizures at the beginning of a heart attack; previous heart attack history of the brain; previous history of diabetes mellitus; previous cerebral infarction in the last three months; previous stroke after waking up; systolic blood pressure > 185 mmHg or diastolic blood pressure > 110 mmHg; blood glucose < 3 mmol/L or more than 22 mmol/L; high risk of hemorrhagic complications; serious complications; taking heparin during the previous 48 hours. The development of fatal intracerebral hemorrhage or symptomatic hemorrhagic transformation of stroke are risky complications of thrombolytic therapy in the acute period of stroke. Asymptomatic hemorrhagic transformation is considered an indicator of reperfusion and may be associated with a favorable clinical outcome. Early use of therapeutic doses of heparin or heparinoids is not recommended in acute ischemic stroke (ESO, 2008). Common indications for heparin prevention after acute ischemic stroke include: high risk of deep vein thrombosis or pulmonary embolism of the lower extremities; cardioembolic ischemic stroke with a high risk of repeated embolization after exclusion of hemorrhagic conversion (atrial fibrillation, artificial valve); replacement of heparin with warfarin is preferable to start taking warfarin immediately rather than switching to it (ESO, 2008); acquired or hereditary coagulation (deficiency of proteins C and S, antiphospholipid syndrome); symptomatic extracranial or intracranial stenosis (stenosis of the internal carotid artery, recurrent TIA or progressive stroke); symptomatic dissection of the extracranial artery. Preventive heparin therapy should be performed only 24 hours after thrombolytic

therapy. In venous stroke, anticoagulant therapy is the main treatment method and should last at least 9-12 months. It should not be used if thrombolytic therapy is planned, in which case aspirin should be prescribed no earlier than 24 hours after the start of thrombolytic therapy (ESO, 2008) [4,9,13].

Patients who do not tolerate aspirin should be prescribed another antiplatelet drug (clopidogrel 75 mg once a day). Neuroprotective therapy. Of the existing drugs with a suspected neuroprotective effect, the largest number of randomized placebo-controlled clinical trials (more than 10) were conducted with seraxone (citicoline). This drug has a high evidence-based neuroprotective activity in the treatment of acute ischemic stroke, and the greatest therapeutic effect is observed when it is used in the first 24 hours. Among the primary neuroprotective measures, intravenous administration of 10-20 ml of magnesium sulfate is often used as a non-competitive NMDA receptor antagonist. At the same time, a multicenter study of intravenous magnesium sulfate administration within 12 hours after the onset of ischemic stroke followed by a 24-hour infusion showed no clinical benefit, with the exception of patients with lacunar infarction. Neuroprotection in ischemic stroke is provided by the use of antioxidants. The mechanism of action of antioxidants is based on the inhibition of non-enzymatic free radical oxidation of lipids, biopolymers, proteins, mucopolysaccharides and nucleic acids, followed by membrane protection, which leads to stabilization of the structural integrity of neurons and preservation of their functional properties. Antioxidants with neuroprotective properties include cavinton, cytoflavin, actovegin, mildronate, and tocopherol acetate (vitamin E). Drugs with pronounced cholinergic and neurotrophic effects (choline alfoselate) and neuropeptides (cortexin, cerebrolysin) are also used. Treatment of stroke complications The most common complications of stroke are cerebral edema, deep vein thrombosis, pulmonary embolism, pneumonia, epileptic seizures, urinary tract infections, and pressure sores. Cerebral edema occurs mainly within 24-72 hours after the onset of ischemic stroke. Pharmacological treatment includes diuretics (furosemide 40 mg two to three times a day) for angioedema and osmotic therapy (25-50 g of mannitol every three to six hours) with interstitial edema of the brain. Hypotonic solutions, glucose-containing fluids, and corticosteroids are not recommended for the development of post-ischemic cerebral edema. In case of stroke, prevention of bronchopneumonia is important. If bronchopneumonia is detected, antibiotics should be prescribed. If seizures recur, antiepileptic drugs are recommended. Treatment of epileptic conditions in stroke is consistent with general principles. Urinary tract infections are a common complication in stroke patients. In most patients, this occurs as a result of prolonged catheterization of the bladder. Therefore, permanent catheters are recommended only in severe cases of stroke. Decubitus prevention includes the use of anti-decubitus mattresses, regular repositioning, skin care, dryness maintenance, and surgical treatment of extensive decubitus. Prevention of vascular complications such as deep vein thrombosis (DVT) and pulmonary embolism (PE) requires preventive treatment using compression underwear and subcutaneous administration of low doses of heparin. In case of DVT or PE, a full dose of heparin should be used. However, heparin therapy not only eliminates the risk of vascular complications, but also increases the risk of secondary hemorrhages. Thus, proper routing of stroke patients and intensive care in a specialized department can significantly reduce the 28-day mortality rate and improve the functional outcome of this disease. However, secondary prevention of stroke is also an important clinical task in order to prevent recurrent cerebrovascular accident. The solution to this problem largely depends on the management of the patient after a stroke. The European Stroke Organization ESO (2008) published the following recommendations for the prevention of recurrent ischemic stroke: all patients with post-ischemic stroke with atrial fibrillation are prescribed oral anticoagulants (warfarin, target INR from -2 to 3, target INR from -2.5 to 3.5 for patients with artificial heart valves). Long-term treatment with oral anticoagulants (warfarin, target value INR-2-3) is indicated for patients with venous stroke, cardioembolic stroke without atrial fibrillation, and patients at high risk of recurrent stroke.; Anticoagulation is not indicated for patients with atherothrombotic ischemic stroke, but it is recommended for patients with repeated TIA or TIA who continue to take anticoagulation. Anticoagulation is not indicated in patients with atherothrombotic ischemic stroke, but it can be used in patients with recurrent TIA or noncardioembolic stroke who are still taking anticoagulation. Anticoagulation should be used to prevent recurrent strokes and recurrent vascular events. It is effective for secondary prevention of ischemic stroke, but in high doses (>150 mg/day. The combination of aspirin and dipyridamole is more effective than aspirin monotherapy for the secondary prevention of ischemic stroke in patients with the atherothrombotic subtype of stroke; if a combination of aspirin and dipyridamole is not possible, the use of low and intermediate doses (50-325 mg/day) of aspirin is the drug of choice to reduce the risk of recurrent strokes. Clopidogrel is somewhat more effective than aspirin in preventing recurrent vascular events, especially in patients with multiple vascular lesions. A combination of diet, exercise, aspirin, statins, and antihypertensive therapy reduces the risk of recurrent stroke by up to 4.8% (86% reduction in overall risk). More aggressive treatment: aspirin plus dipyridamole, antihypertensive drugs, and high-dose statins reduced the risk by up to 2.4% (a 90% reduction in HR). In other words, almost all major cardiovascular events in high-risk groups can be prevented. Unfortunately, today stroke remains the most serious disease and brings huge losses to society. At the same time, the number of cases is growing in working age. At the same time, early detection and adequate treatment in specialized institutions with a focus on the latest achievements of evidence-based medicine is crucial for reducing stroke mortality, restoring lost functions and returning patients to a full life in most cases [7,11,15].

Information about the source of support in the form of grants, equipment, and drugs. The authors did not receive

financial support from manufacturers of medicines and medical equipment.

Conflicts of interest. The authors have no conflicts of interest.

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