

Optimization of Diagnosis and Treatment of Early Neurological Complications in Cardioembolic Stroke

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Abstract Ischemic stroke is a growing medical, social and economic problem that is becoming epidemic in scope. Despite relatively stable incidence rates and declining mortality rates, there has been an increase in the number of life-years adjusted for disability and deaths associated with stroke over the past two decades. Despite the undoubted achievements in this area, the problem of the negative impact of neurological deficits on the effectiveness of rehabilitation measures, short-term and long-term prognosis, functional status and quality of life of patients does not become less significant. Dementia and stroke are often associated with each other, and this combination is associated with higher mortality, poorer recovery, increased readmission rates, and higher economic costs.

Keywords Ischemic stroke, Neuroprotective effect, Cardioembolic stroke

1. Introduction

Currently, several goals are distinguished in the struggle for the survival of brain cells [2]: a decrease in glutamate expression, normalization of ion channels, restoration of phosphatidylcholine levels, and a decrease in the level of arachidonic acid and other inflammatory mediators. The neuroprotective effects of drugs [3] are manifested in an increase in the resistance of brain cells to hypoxia and ischemia; correcting the level of cellular energy; improving blood supply to the brain; increasing the functional activity of neurons and glial cells; normalization of mediator imbalance.

Cardioembolic stroke is one of the most frequent and severe forms of ischemic stroke. In the light of the concept of multifactoriality of the pathogenesis of ischemic stroke, studies of the clinical features of ischemic stroke, including cardioembolic stroke, are highly relevant. The most acute and acute periods are the most important links in the development and course of acute disorders of cerebral circulation.

To objectify the severity of neurological changes and disorders in the functional daily life of patients with stroke in recent years, clinical rating scales have been increasingly used, in particular, NIHSS, Orgogozo, Scandinavian, Guseva-Skvortsova, Bartela [and others]. However, the ambiguity of the data in the literature of such studies in patients in the most acute and acute periods of ischemic stroke, including cardioembolic stroke, is a limiting factor in

the development of qualitatively new rehabilitation schemes. [1]

One of the urgent problems in neurology is the adequate prescription of combined neuroprotective therapy for ischemic stroke (IS) in the acute period, since the result of treatment at this stage largely determines the patient's quality of life after a stroke [5,11].

Acute ischemic damage to neurons during the development of a focus of cerebral infarction (IHM) is based on a complex cascade of interaction between the endothelium of the vascular wall, hemostatic factors, neurons and microglia. Oxygen starvation of tissues stimulates the production of endothelial cells of endothelial dysfunction markers produced by blood vessel endotheliocytes, macrophages, neurons and neuroglia in response to hypoxic brain damage. The so-called "cell death genes" are activated, which are responsible for the development of apoptosis, or programmed death of ischemic penumbra cells, as a result of the expansion of which the infarction volume increases.

With IS, universal patterns of brain tissue response to a decrease in perfusion have been established, which made it possible to formulate a statement about the dynamic nature and potential reversibility of cerebral ischemia and the need to apply urgent measures for the combined restoration of blood flow and protection of the brain from ischemic damage in the acute and acute period in cardioembolic stroke [2,6].

Since each of the neuroprotective drugs has a somewhat limited range of effects on pathological processes in ischemic stroke, it is relevant and substantiated to study the effectiveness of treating patients with a combination of several neurotropic drugs.

Cardiovascular embolism is responsible for the development of 35-40% of all cases of ischemic stroke. The development of cardioembolic stroke is the most formidable complication of cerebral ischemia. Stroke is a qualitatively special condition, being an integrated expression of a complex of complex metabolic, hemodynamic changes occurring in the brain at a certain stage of insufficient blood supply [1,8,9]. The problem of cerebral stroke remains extremely relevant at the present time, which is determined by the significant frequency of its development, the high percentage of disability and mortality of patients [2,10]. Acute ischemic damage to neurons during the development of a focus of cerebral infarction (IHM) is based on a complex cascade of interaction between the endothelium of the vascular wall, hemostatic factors, neurons and microglia. Oxygen starvation of tissues stimulates the production of endothelial cells of endothelial dysfunction markers produced by blood vessel endotheliocytes, macrophages, neurons and neuroglia in response to hypoxic brain damage. The so-called "cell death genes" are activated, which are responsible for the development of apoptosis, or programmed death of ischemic penumbra cells, as a result of the expansion of which the infarction volume increases. In ischemic stroke, universal patterns of brain tissue response to a decrease in perfusion have been established, which made it possible to formulate a statement about the dynamic nature and potential reversibility of cerebral ischemia and the need to apply urgent measures for the combined restoration of blood flow and protection of the brain from ischemic damage in the most acute and acute period in cardioembolic stroke [14,15].

Since each of the neuroprotective drugs has a limited range of effects on pathological processes in ischemic stroke, it is relevant and justified to study the effectiveness of treating patients with a combination of several neurotropic drugs [1,4].

Purpose of the study. Development of new approaches to improving the efficiency of managing patients in the acute period of cardioembolic stroke based on more accurate diagnosis, predicting its course and outcomes, and optimizing neuroprotective therapy.

2. Material and Methods

The study was conducted at the Bukhara Multidisciplinary Medical Center in the Department of Neurology. The study was based on 55 patients with cardioembolic stroke.

Of these, 35 women (63.6%), 20 men (36.4%). The study was conducted at the Bukhara Multidisciplinary Medical Center in the Department of Neurology. All patients underwent a course of treatment for the purpose of neuroprotective therapy, 10 ml of cytoflavin was used (on 5% glucose intravenously once a day, gliatilin 1000 mg intravenously in saline once a day. Cytoflavin and gliatilin were administered in the morning. C) On the 16th day until discharge, the patients continued to receive Cytoflavin.

They received basic therapy at the same time. Basic therapy for cardioembolic stroke included correction of blood pressure, maintenance of normovolemia, and the use of anticoagulants and antiplatelet agents.

3. Result and Discussion

The study was based on the data of a prospective analysis of clinical examination and treatment of 160 patients with cardioembolic stroke. By the method of simple randomization, the patients will be divided into the main and control groups, who received different treatment regimens.

The criteria for the inclusion of patients in the study were: ischemic stroke, acute and acute periods, the ability to perform locomotor functions and neuropsychiatric tests. All patients at the time of examination were conscious and were available for verbal contact. The study was carried out with the consent of the patients and did not contradict generally accepted ethical standards. 85 patients of the main group received basic and combined neuroprotective therapy. Basic therapy for ischemic stroke included correction of blood pressure, maintenance of normovolemia, control of glycemia and body temperature, treatment of cerebral edema and nutritional support, as indicated, the use of anticoagulants and antiplatelet agents. Combined neuroprotective therapy consisted of the use during the first 15 days: Cytoflavin 10 ml (succinic acid 1000 mg, inosine 200 mg, nicotinamide 100 mg, riboflavin sodium mononucleotide 20 mg) on 5% glucose intravenously, once a day; gliatilin 1000 mg intravenously in a saline solution once a day; Actovegin 200 mg intravenously, once a day. Cytoflavin and gliathin were administered in the morning, Actovegin in the second. From the 16th day until the moment of discharge, the patients continued to receive Cytoflavin.

75 patients of the control group received standard treatment, which included a basic therapy similar to the main group, and during the entire hospital period, neuroprotective therapy with ethylmethylhydroxypyridine succinate (Mexidol) or cytoflavin.

Indications for the combined neuroprotective therapy of CEI in the acute period have been developed based on the analysis of its effectiveness.

4. Conclusions

Thus, the use of neuroprotective therapy in the acute period of cardioembolic stroke led to a decrease in disability and mortality in patients. As a result of the study, on the basis of an integrated systematic approach, the concept of the acute period of CEI was developed as a complex system consisting of a complex of synergistic elements, which allows predicting the characteristics of the course, complications and outcomes of the disease, including in conditions of optimization of neuroprotective therapy. Clinical trials have shown that rapid assessment and prompt

treatment can improve clinical outcomes in stroke patients. Early detection of stroke and the use of neuroprotective treatments have improved patients' lives.

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