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OPTIMIZATION OF NEUROPROTECTIVE THERAPY FOR ISCHEMIC STROKE IN THE ACUTE PERIOD

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ANNOTATION

The article demonstrates the world experience of neoroprotective therapy in the treatment of ischemic stroke in the acute perod. The feasibility of prescribing metabolic drugs is not completely clear, the effectiveness has not been definitively proven, despite numerous studies that show only trends. The drug does not reliably improve the neurological status of patients after a stroke, but it reduces the ten-year risk of stroke. Thus, the mechanisms of action of drugs and data from experimental and clinical studies, including those after neoroprotective therapy and with the inclusion of drugs for primary and secondary prevention of ischemic stroke, have been analyzed.

Key words: stroke, ischemic stroke, cerebral infarction, acute cerebrovascular accident, metabolic therapy, neuroprotective therapy, neuroprotective agents, antihypoxants.

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ОПТИМИЗАЦИЯ НЕЙРОПРОТЕКТИВНОЙ ТЕРАПИЯ ИШЕМИЧЕСКОГО ИНСУЛЬТА В ОСТРОМ ПЕРИОДЕ

АННОТАЦИЯ

В статье представлен мировой опыт неоропротекторной терапии в лечении ишемического инсульта при острой периоде. Возможность назначения метаболических препаратов не совсем ясна, эффективность окончательно не доказана, несмотря на многочисленные исследования, которые показывают только тенденции. Препарат достоверно не улучшает неврологический статус пациентов после инсульта, но снижает десятилетний риск инсульта. Таким образом, проанализированы механизмы действия препаратов и данные экспериментальных и клинических исследований, в том числе после неоропротекторной терапии и с включением препаратов для первичной и вторичной профилактики ишемического инсульта.

Ключевые слова: инсульт, ишемический инсульт, инфаркт мозга, острое нарушение мозгового кровообращения, метаболическая терапия, нейропротекторная терапия, нейропротекторные средства, антигипоксанты.

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ЎТКИР ДАВРДА ИШЕМИК ШИКАСТЛАНИШНИНГ НЕЙРОПРОТЕКТИВ ТАЪСИРНИ ОПТИМАЛАШТИРИШ

АННОТАЦИЯ

Мақолада ўткир даврда ишемик қон томирларини даволашда нореопротектив терапиянинг жаҳон тажрибаси келтирилган. Метаболик дори-дармонларни буюриш имконияти тўлиқ аниқ эмас, фақат тенденцияларни кўрсатадиган кўплаб тадқиқотларга қарамай, самарадорлик аниқ тасдиқланмаган. Препарат қон томиридан кейин беморларнинг неврологик ҳолатини сезиларли даражада яхшиламайди, аммо қон томирларининг ўн йиллик хавфини камайтиради. Шундай қилиб, биз дори воситаларининг шу жумладан нореопротектив терапиядан сўнг ва ишемик қон томирларининг бирламчи ва иккиламчи профилактикаси учун дори-дармонларни киритиш орқали таъсир механизмларини ва эксперимантал ва клиник тадқиқотлар маълумотларини таҳлил қилдик.

Калит сўзлар: қон томир, ишемик қон томир, мия инфаркти, ўткир қон томирлари фалокати, метаболик терапия, нейропротектив терапия, нейропротектив воситалар, антигипоксантлар.

The problem of timely neuroprotective therapy of ischemic stroke is the most important in clinical neurology due to its widespread prevalence, high mortality rate, significant disability and social maladjustment of stroke patients. Through research over the past two decades, fundamental approaches to the treatment of ischemic stroke have changed. There were ideas about the readiness of the brain substance to the formation of focal damage, the delay of irreversible changes from the moment of acute cerebrovascular accident (CVA) and the appearance of the first symptoms of the disease, what are determined the attitude towards stroke as a medical emergency requiring rapid and pathogenetic reasonable medical care, preferably during the first 2-3 hours moment of its development [1,6]. Dynamics of mechanisms are launched by acute cerebral ischemia. Deployment dynamics analysis of molecular and biochemical mechanisms is triggered by acute focal cerebral ischemia, established a clear time sequence of their "Inclusions".

It was found that the degree of the damaging effect of ischemia is determined primarily by the depth and duration of the decrease in cerebral blood flow. The area of the brain with the most pronounced oligemia (less than 10-15 ml) becomes irreversibly damaged very quickly, within 6-8 min.

The moment of development of ischemia ("core", or "nuclear" zone of ischemia), energy metabolism and there are only functional, but not structural changes. Within a few hours, the central "point" infarction is surrounded by ischemic, but living tissue - the zone of "ischemic penumbra", or penumbra, in which, as a whole, energy metabolism and there are only functional, but not structural changes. Region "Ischemic penumbra" can be saved by the restoration of adequate perfusion of brain tissue and application neuroprotective agents. Exactly penumbra is the main target stroke therapy in the first hours and days of diseases [4,8]. The duration of the existence of the penumbra is individual for everyone, the patient and determines the boundaries of the time period, within which with most effective can carry out medical measures. Stroke ranks first among the causes of persistent disability, which necessitates timely drug therapy not only in the acute period of the disease, but also throughout the recovery phase.

The development of acute cerebral ischemia triggers pathobiochemical cascade reactions, the outcome of which is a cerebral infarction (MI), which is formed by two mechanisms: necrotic cell death and apoptosis - programmed cell death (Skvortsova, V.I., 2001). These modern pathogenetic concepts allowed us to propose a scheme the sequence of stages of the "ischemic cascade" on the basis of their causal relationships. (U. Dirnagl, C. Iadecola, M. A. Moskowilz, 1999). Each stage of the cascade is a target for the therapeutic effect of drugs, primarily with neuroprotective effects. The earlier the cascade is interrupted, the greater the effect can be expected from therapy (Lee R.G., van Donkelaar P., 1995; Lutser H.L., Clark W.M., 1999; Lees K.R., 2000, Skvortsova V.I., 2004).

Among all strokes, the proportion of ischemic strokes is, about 80% (81.3% for men and 82.3% for women in 2010). According to the generally accepted TOAST classification, there are five pathogenetic subtypes of AI:

1. atherothromboembolic;
2. cardioembolic;
3. lacunar (due to occlusion of a small vessel);
4. stroke of another established etiology;
5. stroke of unknown etiology.

With the duration of symptoms less than 24 hours and the absence of cerebral infarction, according to neuroimaging data, the diagnosis is formulated as a transient ischemic attack (TIA). Risk factors for the development of ischemic stroke are usually divided into modifiable and non-modifiable. Non-modifiables include old age, male sex, hereditary burden, low birth weight (less than 2500 g) and race. Arterial hypertension, hyperlipidemia, atherosclerosis, smoking (passive and active) [9, 10], ischemic heart disease, cardiac arrhythmias, valvular and other heart defects are currently considered as the main modifiable risk factors for ischemic stroke, sedentary lifestyle, obesity, diabetes mellitus, alcohol abuse.

Among the predictors or risk factors for stroke, migraine with aura, sleep apnea, erectile dysfunction, hyperhomocysteinemia, oral contraceptives, antipsychotics, corticosteroids, blood diseases, hypercoagulation, various vasculitis, lesions of arterial and venous vessels of the lower extremities are also distinguished [9]. Social and economic factors influence mortality: mortality is higher at a lower income level [2, 3]. The maximum mortality (about 30%) is observed in the first 28 days after stroke [3, 6, 9]. Therefore, therapeutic measures and prevention of recurrent stroke should be started as early as possible and carried out as actively as possible in the acute period of stroke. However, even after the patient has passed the four-week line and the risk of an unfavorable outcome has significantly decreased, treatment should not be stopped. The main areas of patient management after a stroke include the prevention of repeated vascular catastrophe and active rehabilitation aimed at compensating for lost skills (motor, speech, etc.), social and psychological assistance.

Treatment. There are medicinal, non-drug and surgical methods of treatment and secondary prevention of stroke. In the acute period of ischemic stroke, treatment includes basic therapy: control and maintenance of vital functions (respiration, blood circulation, water and electrolyte balance), correction of glycemic levels, blood pressure, and decongestant therapy [3, 5]. After excluding the hemorrhagic nature of cerebrovascular accident, a specific therapy is performed using computed tomography. The most effective is thrombolytic therapy aimed at dissolving or removing a thrombus (or embolus) in an occluded vessel. It can be carried out within the first 4.5 hours ("therapeutic window") from the moment of the onset of neurological symptoms [3, 9, 10]. After a short clinical, laboratory and instrumental examination, in the absence of contraindications, a tissue plasminogen activator is administered intravenously to the patient in an intensive care unit. Then, within 24 hours, the patient is under close supervision of medical personnel, he also undergoes repeated computed tomography [9,10].

If a patient with ischemic stroke does not have time to get to the hospital during the "therapeutic window", then specific therapy begins with antiplatelet drugs that reduce platelet aggregation, prevent their fixation on the atherosclerotic plaque and resist the formation of thrombosis. In the acute period of ischemic stroke, the most studied is the effectiveness of acetylsalicylic acid 160–325 mg / day [9]. After 48 hours, other antiplatelet agents (clopidogrel 75 mg / day, a combination of acetylsalicylic acid 25 mg and sustained-release dipyridamole 200 mg) may be prescribed to patients with noncardioembolic stroke. According to the results of several large studies, there was a higher antiplatelet activity of clopidogrel or a combination of acetylsalicylic acid and dipyridamole compared with monotherapy with acetylsalicylic acid [9]. It has been proven that long-term use of antiplatelet agents in patients who have undergone ischemic stroke significantly reduces the risk of further acute cerebrovascular accidents [3, 7, 8].

With the cardioembolic nature of IS against the background of atrial fibrillation, valvular defects, etc., anticoagulants are used. The greatest practical experience has been gained with the use of warfarin. However, the use of warfarin is associated with some inconveniences (the need to monitor the international normalized ratio at least twice a month) [6,9,10]. Therefore, in nonvalvular atrial fibrillation, preference is given to direct oral anticoagulants, which do not require regular monitoring of the international normalized ratio. These drugs (dabigatran, rivaroxaban, apixaban) are as effective as warfarin and have a similar safety level.

Rehabilitation measures. The goal of neurorehabilitation of patients who underwent IS is to restore or replenish lost neurological functions and provide self-care ability, which largely depends on the neurological defect in a particular patient. For example, with isolated mild hemiparesis or isolated hemihypesthesia, patients can go unaided most of the time. With the development of hemianopsia, the patient may require outside help when moving. The prognosis significantly worsens with partial or complete immobility of the patient, as well as with the development of severe cognitive impairment, speech disorders, anosognosia, depression, severe pain syndrome, gross bulbar or pseudobulbar disorders. Early motor activation is of great importance

for faster and more complete recovery and compensation of neurological functions [7]. Regular walking will gradually improve your gait. Special gymnastics and training in performing normal household activities increase the chances of a more complete recovery of upper limb function. In case of severe spasticity, which complicates neurorehabilitation, muscle relaxants are used, including local injections of botulinum toxin; in case of foot drooping due to paresis, the use of an orthosis is recommended. Movement rehabilitation should be aimed primarily at restoring daily household skills (dressing, toilet, cooking and eating, moving around the house and outdoors) in order to achieve maximum patient independence. To reduce the severity of speech disorders, speech therapy classes are highly effective. With the development of post-stroke cognitive impairment, the patient's prognosis in terms of rehabilitation, quality and life expectancy significantly deteriorates, and the patient's adherence to therapy also decreases [2,3]. It has been shown that during the first year after a stroke, 25-30% of patients develop dementia, while in one third of them it becomes the result of decompensation of the concomitant neurodegenerative process [4,5].

In post-stroke dementia, acetylcholinergic (donepizil, galantamine, rivastigmine) and glutamatergic (memantine) drugs are used, which lead to a decrease in cognitive and other neuropsychiatric disorders. Acetylcholinesterase inhibitors partially compensate for the acetylcholinergic deficit that occurs against the background of damage to the central acetylcholinergic structures, and memantine normalizes the processes of impulse transmission in glutamatergic synapses. The choice of a specific drug is determined by the severity and nature of cognitive impairment, concomitant diseases, and drug tolerance. It is important to combine pharmacotherapy and non-pharmacological methods. There are methods of individual and group sessions (cognitive training, cognitive stimulation) that slow down the progression of a cognitive defect and compensate for it [2,5].

Emotional and behavioral disorders (in particular, vascular depression), which increase the risk of recurrent stroke and other vascular events, and cause or enhance cognitive impairment, are one of the important disorders that are often ignored by doctors and relatives [6]. Patients who have suffered a stroke and have depression are recommended psychotherapy and pharmacotherapy with antidepressants [6, 9].

Neuroprotective and neurometabolic therapy is designed to reduce damage caused by pathological processes occurring in the brain substance during ischemic damage, to activate the processes of neuroplasticity and neuroreparation [7,9, 11]. For this, drugs such as Cytoflavin, piracetam, choline alfoscerate, vinpocetine, citicoline, and standard ginkgo biloba extract are widely used.

Conclusion. Patient management in the acute and early recovery periods should be comprehensive and include non-drug and medical methods. Antihypertensive, antiplatelet or anticoagulant, lipid-lowering therapy should be carried out, taking into account the individual characteristics of the patient. The combination of several means of secondary prevention of ischemic stroke can reduce the risk of recurrent stroke by almost 80%, thereby preventing four out of five possible strokes [7].

Neuroprotective and neurometabolic therapy plays an important role both in the acute and in the recovery period of stroke, which contributes to a faster and more complete recovery of impaired neurological functions, provides pharmacological support for neurorehabilitation activities carried out simultaneously. The patient and his relatives should be explained the purpose and significance of not only medication, but also non-medicinal methods, as well as the importance of all measures in the recovery and subsequent periods.

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