NEUROPROTECTIVE APPROACH TO TREATMENT OPTICORETINAL PATHOLOGY

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https://doi.org/10.5281/zenodo.10119120

Abstract. *The term "neuroprotection" came to ophthalmology from neurology. Neuroprotective therapy — Events, directed on prevent- rotation of the cascade of reactions leading to damage to neurons and caused by nyh, mainly, ischemia. We are talking about sequentially developing reactions, when inpathological process involves more and more new neurons, and surrounding the affected tissues stinging them themselves become a source of pathological influences. In this regard, neuroprotective treatment should be carried out in within the so-called "therapeutic window", when the defeat of the nervous fabrics more gone irreversible.*

Keywords: *the pathogenesis of optic neuropathies is characterized by the appearance of cells of the network chatki, which are in a state of parabiosis (from the Greek para - next to, around and bios - life).*

At the heart of the phenomenon of parabiosis of excitable tissues (primarily th, nervous) lies the lack of energy production by the cell, which is the first turn disrupts the active transport of electrolytes across the membrane. WHO- there is no permanent depolarization of the membrane: the cell lives, but does not function etc. The cessation of pathological influences leads to the restoration of the original th functional state of the cell, and their further impact - to its death, which in many ways defines progression diseases. In pathological processes with a primary lesion of the visual of the nerve, the axons of the GCS (white matter of the nervous tissue) are primarily affected neither), and only after a certain time, the corresponding "therapeutic mu window, irreversibly are amazed ganglionic cells (grey substance nervous tissue), which creates the prerequisites for the successful use of neuro- protective drugs.

Among major reasons leading To death GKS, allocate:

neurotrophin deficiency due to blockadetrograde axonal transport;

glutamate excitotoxicity ;

free radical damage ;

neurotoxicity, related With oxide nitrogen ;

apoptosis .

Neurotrophin deficiency. Neurotrophins are a family of re- gulatory proteins nervous fabrics, which synthesized neurons And cell-kami glia, promote proliferation, differentiation, and support zania vitality and functioning neurons. Neurotrophins relyse effects several ways, main from which (before 70 %) is-etsya retrograde. Retrograde axonal transport moves neutrophins from the brain to the GCS, thus ensuring their survival. Wednesdaydineutrophins secrete: BDNF (brain-derived neurotrophic factor) - neuro-trophic factor, isolated from brain, NGF (nerve growth factor) — factor growth nerves NT-3 (neurotrophin-3) — neurotrophin 3 And NT-4/5 (neurotrophin-4/5) — neurotrophin 4/5. Broadcast visual signal from GKS Vthe brain is possible only when growth factors are delivered from the brain to the GCS [11]. Atblockade retrograde axonal transport admission neurotrophinsTo GKS decreases What Maybe contribute their death.

Glutamate excitotoxicity. Excitotoxicity (from Englishexcitotoxicity - toxicity that develops when excited) - starting mechanism of necrotic and apoptotic neuronal death with multiple gih neurodegenerative violations. Glutamate excitotoxicity arises at promotion concentration extracellular glutamate.

Glutamate is the main excitatory neurotransmitter in the CNS ry is present in neurons in significant concentrations. High con- concentration of glutamate causes the activation of several types of cellular receptors, including NMDA receptors (N-methyl-DL-aspartate). As a result this is an increase in Ca2+ concentration in cells and accumulation K+ ions in the extracellular space. "Calcium overload" of neurons And activation Ca2+-dependent processes (increase activity proteases kinases, endonucleases, lipoxygenases, phospholipase A2 and other enzymes) leads to significant changes in metabolism and genetic apparatus cells, uncontrolled action free radicals And Maybe You- call for irreversible cellular death.

Free radical damage. The appearance of free radicals fishing is possible not only through the activation of glutamate-calcium excito- sity, but also within the normal oxidative activity of tissues, especially especially with a high metabolic rate. These structures include networks chat. Inactivation free radicals carried out endogenous antioxidants — superoxide dismutase, vitamins E and WITH, glutate- ion. In case of insufficiency of antioxidant mechanisms, toxiccial damage to cellular structures, which is realized in damage to proteins kov molecules, nucleic acids And V peroxide oxidation lipids.

Neurotoxicity associated with nitric oxide. Neurotoxic Effect oxide nitrogen arises due to his reactions with superoxide- anion and formation peroxynitrite, which is strong oxidizing agent. Due to its properties, it is capable of causing damage of a wide range of molecules in the cell, including DNA and proteins. Per- oxynitrite diffuses along the axons deep into the retina, also causing apoptosis GK, A spreading peroxynitrite by direction to brainleads to loss cells in outdoor knee bodies.

Apoptosis. Apoptosis (from the Greek apoptosis - falling off) is the phenomenon programmed cell death, accompanied by a set of characteristic cytological signs (markers apoptosis) and molecular processes.

Thus, during the development of any of the considered optical neuropathy V result impact on axons GKS certain pathological factor, the death of HA does not occur immediately. Damage first axons of the GCS lengthens the period of the "therapeutic window", when the cell is in balance sits between death and restoration and creates background for successful neuroprotective treatment [12].

It is customary to distinguish two groups of neuroprotective drugs - direct my and indirect neuroprotectors.

Direct neuroprotectors directly protect the neurons of the network chats and fibers visual nerve. These drugs block main cell damage factors that are caused by the development in this zone ischemia, as a result of which there is an increase in the concentration of pro- ducts of lipid peroxidation, free radicals, Ca2+ ions, acidosis.

Direct neuroprotective properties have:

- natural vitamins And flavonoids: vitamins A, E, WITH, GABA;
- enzymes antioxidant systems body: superoxide dismuaza;
- non-enzymatic antioxidants: emoxipin, mexidol And histochrome;
- blockers calcium channels: betaxolol, latanoprost, nifedi-

− neuropeptides: retinalamin, cortexin; antihypoxants — cytochrome WITH;
− alpha agonists: brimonidine.

Currently, a search is underway for drugs that could to eliminate the factors contributing to the activation of apoptosis. Day- The action of these drugs is aimed at reducing the adverse effects glutamate And others substrates on axons ganglion neurons

Indirect neuroprotective action implies the effect of pre- paraty to various factors that increase the risk of cell damage (decrease in perfusion pressure, atherosclerosis, changes in rheologicalblood properties, angiospasm), as well as increasing the body's resistance to decrease in oxygen perfusion pressure in tissues. Similar effect volume is possessed by drugs that improve microcirculation (theophylline eti- lendiamine and nicotinate, nicergoline, vinpocetine, pentoxifylline, etc.), rheological properties of the blood, reducing the level of cholesterol in the blood, nootropic agents (cerebrolysin, citicoline).

Many years of experience in neuro- and retinoprotective treatment of various optic neuropathy convinced us that one of the most effective neuroprotective drugs is a nootropic drug citicoline groups. It has a wide spectrum of action, affects all pathological links development apoptosis GKS: 1) slows down process destroy-membranes of ischemic neurons by inhibiting the activity phospholipase A2, restores their structure and function by stimulating tions biosynthesis of phosphatidylcholine one of structural glue elements accurate membranes (R. M. Adibhatla et al., 2002); 2) reduces release glutamateinhibits transmembrane transport of calcium ions into the cell (O. Hurtado et al., 2005); 3) normalizes the energy of mitochondria, restores the function cationization of Na + / K + -ATPase, a reduced level of ATP in the brain tissue, energy processes V neurons (A. A. Farooqui et al., 2000;

J. J. Secades, 2002); 4) reduces oxidative stress after ischemia/ reperfu-zii, raises activity endogenous antioxidant systems protection gluecurrent due to stimulation of the synthesis of glutathione - a non-enzymatic factor of internal recellular antioxidant protection and increase enzyme activity glutathione reductase (RM Adibhatla et al., 2002); 5) prevent death neurons through suppression expression proteins, participating V developmentapoptosis after ischemia (J. Krupinski et al., 2002; C. Mir et al., 2003).

Given the fact that glaucoma should be considered a neurodegenerative disease, anterior ischemic optic neuropathy is aessence ischemic stroke of the optic nerve (part of the brain, hay on periphery), A neuritic optic neuropathy — result compression of the optic nerve by inflammatory exudate with slowing down more blockade of the axoplasmic current at the level of demyelinating or infectious lesions, we compared the effectiveness of the use of cyto- Colin in the treatment of patients with glaucoma, ischemic and neuritis ticoneuropathies [6]. As a result of the study, we made the following conclusions: 1) citicoline has a pronounced neuroprotective effectwith optic neuropathies of various origins, increases visometric and rimetric functional indicators and subjective assessment of the quality vision of patients; 2) the most pronounced neuroprotective effectciticoline has an effect on neuritic optic neuropathy, which can be clarify less significant defeat axons GKS at optical neuritis compared with ischemic and degenerative processes in visual nerve; 3) V some cases appointment citicoline leads To subjective improvement in the quality of vision in patients with optic neuropathyin the absence of positive dynamics of visometric and perimetric ric indicators.

In the treatment of optic neuropathies of various origins, the greatest effectrenders sub-Tenon way introductions citicoline (Ceraxon, Cera- lin - 2 ml: 1 ml in the upper fornix, 1 ml in

the lower fornix). If this is not possible, we administer the drug intravenously (1000 mg 10 injections daily or through day) with subsequent lengthy reception his tableted forms (Strocyte 500 or 1000 mg 2 times a day for 2-3 months). With absence possibilities and intravenous administration of citicoline, we recommend a long body reception maximum doses tableted forms drug — Strocyte 1000 mg 2 times a day for 3 months. We offer a repeated course through six months in the absence of urgent indications for its earlier appointment cheniya.

Thus, studies of the pathogenetic features of the development studies of various optic neuropathies have shown similar mechanisms of damage nervous tissue. The optic nerve, which is the axons of the GCS, is affected primarily, retinal neurons are affected secondarily. With varioustypes of optic neuropathies GCS are affected to varying degrees and in different ways react for neuroprotective treatment.

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