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MEDICAL SCIENCE

Risk factors affecting cerebral circulation disorders

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In patients with arterial hypertension, destructive changes occur in the walls of cerebral vessels, mainly in the muscular wall of arterioles: the ion permeability of smooth muscle cell membranes increases, followed by their hypoxia, anoxia and death. The number of smooth muscle cells in the muscular membrane of the proximal, distal lenticulostriate arteries, enveloping cortical arterioles decreases by more than 50% of the initial number. These changes lead to diffuse accumulation of cell decay products in the vascular wall, fibrinoid necrosis, formation of microaneurysms in arterioles with a diameter of 70-700 microns, which are rupture sites [3].

Of great importance in the development of non-traumatic intracerebral hemorrhages are the features of the blood supply to the subcortical nodes: the lenticulostriate arteries depart from the middle cerebral artery at a right angle, the number of anastomoses between them is small, which does not provide sufficient depreciation in hypertensive cerebral crises and leads to their rupture [3].

Currently, there are two leading mechanisms for the development of stroke intracerebral hemorrhages: rupture of a pathologically altered vessel with the formation of a hematoma and hemorrhagic impregnation. Hemorrhages in the form of a hematoma, according to autopsy materials, are found in 85% of cases, have typical localization, are located in the subcortical nodes, in the lobes of the brain (lobar) and in the cerebellum. In the acute period of the disease, with this type of hemorrhage, there is no significant destruction of the brain substance, since the outflowing blood pushes the nerve fibers apart. In 15% of cases, hemorrhagic impregnation of the brain substance develops, which most often develops in the thalamus, in the pons and is accompanied by significant destruction of the surrounding brain tissue [1].

The formation of intracerebral hematomas leads to vascular-reflex, parabiotic and necrotic reactions in the brain tissue. The volume of hemorrhage in most cases increases within 2-3 hours after the disease. Subsequently, local and

distant vascular reflex reactions develop, leading to a cascade of pathophysiological reactions in brain structures, to their ischemia, edema, and swelling [2].

However, to date, based on an understanding of the pathobiochemistry of ischemia, the study of the neuroprotective effects of drugs that interfere with the mechanisms of excitotoxicity, the development of oxidative stress, as well as drugs with neurotrophic action is considered a promising direction. (5). And, despite the presence of many unresolved problems and contradictions regarding the drug therapy of stroke, an increase in its effectiveness in the appointment of patients with various drugs with different mechanisms of action, points of application and their combination, aimed at correcting pathological processes in stroke, both for theoretical and and for practical medicine is an urgent problem. One of the reasons for severe disability and high mortality in patients with hemorrhagic stroke is the development of recurrent hemorrhage.

Among patients with cerebrovascular pathology suffering from epilepsy, in 27% of cases, this disease was associated with a previous stroke, in the rest - with signs of CCI, manifested by "silent" strokes in the blood supply basin of the predominantly middle cerebral artery, hypodense foci of hemispheric localization.

Vascular diseases of the brain are one of the most important problems of modern neurology and cardiology. Increasing prevalence, disability, high mortality of patients with very limited prospects for the restoration of impaired functions and ability to work determine the medical and social nature of their consequences [1].

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