HISTOTOPOGRAPHIC CHANGES IN THE WALL OF CAPILLARY BLOOD VESSELS IN EXPERIMENTAL SUBARACHNOID HEMORRHAGE

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Abstract. The content of the article is devoted to the study of histotopographic changes in capillary vessels during autogenous subarachnoid hemorrhage of the brain in dogs under experimental conditions. Spasm of the main arteries of the brain, especially spasm of arterioles directly involved in the vascularization of the brain, in turn, causes disruption of cerebral blood circulation.

Keywords: experiment, dog, arachnoid membrane of the brain, hemorrhage, main vessels, capillaries, autogenous blood, endothelial cell.

ГИСТОТОПОГРАФИЧЕСКИЕ ИЗМЕНЕНИЯ СТЕНКИ КАПИЛЛЯРНЫХ СОСУДОВ ПРИ ЭКСПЕРИМЕНТАЛЬНОМ СУБАРАХНОИДАЛЬНОМ КРОВОИЗЛИЯНИИ

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Аннотация. Содержание статьи посвящено изучению гистотопографических изменений капиллярных сосудов при аутогенном субарахноидальном кровоизлиянии головного мозга у собак в экспериментальных условиях. Спазм магистральных артерий мозга, в особенности спазм артериол,

непосредственно участвующих в васкуляризации головного мозга, является, в свою очередь, причиной нарушения мозгового кровообращения.

Ключевые слова: эксперимент, собака, паутинная оболочка мозга, кровоизлияние, магистральные сосуды, капилляры, аутогенная кровь, эндотелиальная клетка.

Introduction. Vascular spasm and secondary ischemic changes in the brain during aneurysm ruptures are among the most severe complications of non-traumatic subarachnoid hemorrhage [1,2,3]. Given the high prevalence of cerebrovascular pathology, it is necessary to analyze the ultrastructure of the main arteries and arterioles of the brain's pia mater in a model of subarachnoid hemorrhage [4,5] accompanied by arterial spasm, particularly in the stage of prolonged arterial spasm, which leads to the development of ischemia, brain edema, damage to nerve cells, and impaired cerebral circulation [6,7,8].

Study Objective. To investigate the histotopographic changes in the walls of capillary vessels in experimental subarachnoid hemorrhage.

Materials and methods. The ultrastructure of the main arteries and arterioles of the pia mater (terminal branches of the main arteries directly involved in brain vascularization) in dogs was studied on days 3–5 after the injection of autologous blood into the cerebrospinal fluid.

Results. Three days after the injection of autologous blood into the cerebrospinal fluid, an increase in the elements of the granular cytoplasmic reticulum, free ribosomes and polysomes, mitochondria, and lysosomes was observed in the cytoplasm of endothelial cells of the arteries. The nuclei of the cells often assumed an irregular oval shape. Rounding and gradual isolation of endothelial cells led to a significant expansion of intercellular spaces. In the adventitia, single erythrocytes or groups of 2–3 macrophages were detected. Throughout the media, areas of myofibril rarefaction were found in the sarcoplasm of smooth muscle cells, both in the superficial layers of myocytes facing the adventitia and, in the layers adjacent to the intima. Areas of myofibril rarefaction were also observed in the perinuclear zone and peripheral parts of the cells; these areas were irregularly oval in shape,

with sizes ranging from 100 to 30 nm. Within these areas, unstructured osmiophilic formations of oval shape were sometimes detected. In addition, an increase in the number of elements of the granular cytoplasmic reticulum, free ribosomes and polysomes, and the appearance of lysosomes were noted in the sarcoplasm of myocytes. Against the background of changes in the adventitia and media, the internal elastic membrane of the arteries became folded, and the shape and orientation of endothelial cells changed. The long axis of the cells was often oriented radially relative to the arterial lumen. The length of cytoplasmic protrusions into the vessel lumen ranged from 250 to 4200 nm, with a width of 100–2800 nm. The protrusion of the cytoplasm of endothelial cells could contain a nucleus of irregular oval or elongated shape. The shape of endothelial cells varied: rounded, hourglass-shaped, or racket-shaped. Over significant lengths, the arterial lumen could be divided by radially oriented cells into “bays.” Depressions in the plasma on the surface of endothelial cells measured 500–1300 nm. The cytoplasm of the cells showed an increase in the number of free ribosomes, polysomes, elements of the granular cytoplasmic reticulum, and lysosomes. Changes in the ultrastructure of smooth muscle cells, the configuration of the internal elastic membrane, and endothelial cells of the intima of the pia mater arterioles in dogs generally corresponded to those in the main arteries. Areas of myofibril rarefaction were also found in the sarcoplasm of myocytes; the internal elastic membrane was folded, fragments of endothelial cells protruded into the vessel lumen, and “bays” were present between the cytoplasmic protrusions. Previously, under similar experimental conditions, when studying the ultrastructure of paravasal nerve trunks of the external (superficial) nerve plexus of the main arteries of the dog brain, we identified changes in the ultrastructure of the endothelium, perineurium of nerve trunks, axons of myelinated nerve fibers (appearance of gaps in the myelin sheath, agglomeration of neurofilaments, mitochondrial edema), and, to a lesser extent, axons of unmyelinated nerve fibers (uneven distribution of neurofilaments, mitochondrial edema). The system of brain membranes represents a system of paracerebral barriers, in which we identified a structural-functional group of

cerebrospinal fluid-tissue barriers (CSTB), which includes, in particular: the cerebrospinal fluid-muscular barrier (CSMB), whose morphological substrate is the adventitia of the main arteries as a whole; the barrier between cerebrospinal fluid and smooth muscle cells of the pia mater arterioles (CSMB), whose morphological substrate consists of the components of this layer; the cerebrospinal fluid-neural barrier (CSNB), whose morphological substrate includes the endothelium and elements of the peri- and endoneurium of paravasal nerve trunks of the main arteries of the brain. It has been shown that the etiological factors of arterial spasm developing in subarachnoid hemorrhage in patients and in its modeling are primarily the effects on the walls of cerebral arteries located in the cerebrospinal fluid-carrying channels of the leptomeninges, serotonin, and fibrinolysis products. Under the conditions of the identified disruption of the integrity of cerebrospinal fluid-tissue barriers, intensive penetration of spasmogenic factors with cerebrospinal fluid into the deeper layers of the walls of arteries, arterioles, and paravasal nerve trunks becomes possible, with subsequent damage to smooth muscle cells and axons.

Conclusion. The described disruption of the integrity of the morphological substrates of CSMB, CSMB, and CSNB represents important pathogenetic links in the development of arterio-arteriolar spasm. Spasm of the main cerebral arteries, especially spasm of arterioles directly involved in brain vascularization, in turn, causes impaired cerebral circulation.

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