International Journal of Psychosocial Rehabilitation, Vol. 24, Special Issue 1, 2020
ISSN: 1475-7192

Cortical Morphological Characterization of the Cerebellar Stingray Slice in Amputation

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Abstract--*In this paper, the aim is to develop a specific morphological change of the cerebellar cortical stingray slice during amputation. At amputation of the limb, a characteristic complex of features is determined in early terms: acute neuronal swelling, increase of perivascular expansion of spaces, pericellular expansion of spaces and hyperoxyphilia of microglia. These morphological features may cause ischemic changes in neurons. In the ganglion layer, there is a pronounced swelling of Purkinje cells with a widespread loss of astrocytes in the molecular layer of the cortex of the cerebellum stingray lobes due to tissue hypoxia, which may be reflected in the form of cariopicnosis and caryorexis nuclei.*

Keywords--*trauma, leg amputation, cerebellum, histology, stingray slice, cerebellum cortex.*

I. INTRODUCTION

The relevance of the problem. Amputation of the extremities is one of the most frequent types of trauma. According to WHO, in technically developed countries fractures of bones account for 5 to 10% of all violent deaths [1]. Mortality from injury has tended to increase in recent years. Globally, between 60 000 and 80, 000 people die annually from bone fractures, with 90% of these deaths occurring in older and older persons [2]. In the Russian Federation, this type of death accounts for about 1% of all forensic autopsies, according to reported data from consolidated territorial forensic medical examination bureaux. If fractures of bones did not cause death at the scene, morphological changes in the victim's body would develop in a specific pathogenetic sequence. The combination of these features is called pathomorphology of traumatic disease. The state of the central nervous system (CNS) plays a vital role in the implementation of all life processes. Therefore, the attempt to establish the criteria of lifetime and age of bone traumas based on the study of morphological changes of the CNS in this pathological state is relevant [3-10]. It causes the importance of developing new methods that expand the possibility of expert evaluation of bone injuries.

The aim of the study is to develop the peculiarities of morphological changes of the cerebellar cortex stingray slice during limb amputation.

Research material and methods

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DOI: 10.37200/IJPR/V24SP1/PR201174
Received: 16 Jan 2020 | Revised: 06 Feb 2020 | Accepted: 25 Feb 2020

We studied the condition of a slice of cerebellar cortex stingray from 7 days to 90 days from the moment of limb amputation. We used routine histological staining with hematoxylin and eosin and Nissl. Immediately before fixation of the tissue, the cerebellum was cut out in the form of 0.5×1.0×1.5 cm pieces and placed in 10% formalin solution for 1 day. Histological preparations were prepared by pouring pieces of internal organs into paraffin, followed by slices 5-7 microns thick, colored with hematoxylin and eosin and Nissl. Morphological visualization of changes was carried out on an optical microscope adapted with the computer system of image analysis.

II. RESULTS AND DISCUSSION

Microscopic picture of the cortical lobes of the cerebellum stingray on day 7 of the study: the lumen of small arteries slightly dilated with spasms. Small arterioles in most fields of vision with falling walls. The perivascular spaces in these vessels are not dilated. In the capillaries, there is irregular blood filling with a predominance of anemia, areas of stasis and erythrocyte slagging. Zones with slotted enlargement of pericapillary spaces at small intervals were fixed. Areas of lumen enlargement were determined in veins during the whole period, and in a number of vision fields - erythrocyte sliding in vessel lumen. Uneven wall swelling and perivascular expansion of spaces in single fields of vision (Fig. 1).

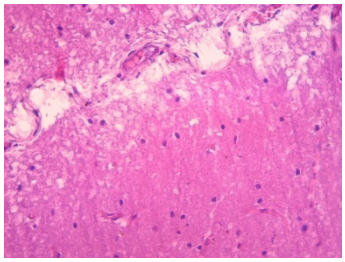

fig. 1. Vessel fullness, perivascular swelling.

A slight swelling of conductive fibers was observed in white matter. In the molecular layer there was a slight swelling of basket neurons and protoplasmic astrocytes, in the ganglion layer - a weakly expressed swelling of single astrocytes, Purkinje's cells (Fig. 2) without loss of ramifications and cell deposition, in the granular layer - preservation of histo-architectonics. In single cells of fine clay there is a slight expansion of pericellular spaces. Sometimes pigmentation and lipoidosis of nerve cells were noted. In white matter of the brain, dilated venules containing red blood cells were detected, which were visible in single fields of vision in perivascular spaces.

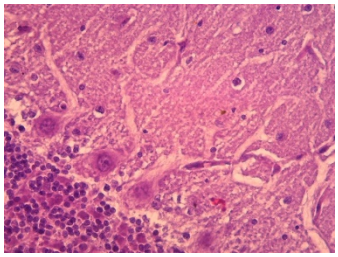

fig. 2 Pericellular oedema of Purkinje cells without loss of astrocytes.

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Light-optical microscopy of cerebellar stingray cortex slices 30 day study: uneven expansion of vessels; widespread expansion of perivascular spaces, mainly in the molecular layer of the cerebellum cortex. In the cerebellum cortex - cells with slightly reduced content of Nissl. Changes in pear-shaped neurons by the type of acute swelling were determined. In brain matter, there was no expansion of perivascular spaces, arterioles became wave-like. In the lumen, arterioles were small erythrocytes. Capillaries were narrowed along the whole length, venules were expanded and overflowed with blood form elements with fine fibrin threads, there were no optically empty spaces around. We observed flattening of endothelial cells in vessel walls and swelling of single periadventitial cells. In perivascular glia - expansion of pericellular spaces. In cytoplasm of single neurons large and small vacuoles were found, which is a sign of severe dystrophy; single neurons with signs of hyperchromatosis and cariopicnosis, reflecting the full depth and severity of the destructive process with the increase of perivascular hemorrhages in white matter.

In glial cells of the molecular layer, the necrobiotic process and spontaneous white matter, vacuumization were observed (Fig. 3). Expansion of pericellular spaces in small clay cells, basket neurons and protoplasmic astrocytes. Vacuumization with hyperchromatosis of cells of the molecular layer was noted. In the ganglion layer - moderate swelling of Purkinje cells without loss of spurs and cell deposition, swelling of single astrocytes. There are areas with sharply swollen shoots alternating with areas without swollen shoots. We noticed sharp swelling of Purkinje cells with cariolysis of single neurons. The number of nucleus organizers in the cytoplasm of ganglionary astrocytes decreased. There are signs of fragmentation in single astrocytes of this layer.

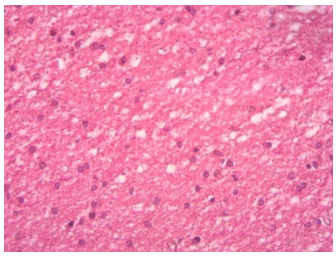

fig. 3 Oedema, spongiliolysis of the molecular layer of the cerebellar cortex.

By the 90th day, when colored by Nissl, cerebellar cortex neurons had different degrees of basophilia: light and dark cells were constantly determined among them. In the field of vision of a light microscope, relatively large light cells had a large, round, swollen light nucleus; their cytoplasm was weakly basophilic. The Nissl substance was finely dispersed in its structure, consisting of a small number of blocks. The neurons had a rounded or irregularly shaped nucleus with a central nucleus. In cytoplasm, diffuse basophilia. Perivascular and pericellular edema increased, neuronal changes in the cerebellum cortex progressed. The main sign of severe changes in nerve cells during this period was a major disturbance of the structure of all the main elements of the cell: the cytoplasm, nucleus and nucleus. In these terms, more frequent were cells that took irregular shape and fuzzy contours, their ramifications were thinning, and a light belt was formed around the cells. The nuclei and the nucleus of a number of cells could not be distinguished. In cells of fine clay, basket neurons and protoplasmic astrocytes there was a significant expansion of pericellular spaces.

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In cerebellar cortex, a sharp full-blooded venous channel was observed without erythrocytes entering the perivascular spaces. In the arteries - wall paresis with slight mild swelling of endothelial cells. We observed foci with insignificant wave-like movement of vessel walls; perivascular expansion of spaces. In a number of fields of vision we revealed plasma registration in the lumen of the vessels. There were centers of anaemia of capillaries. Leukostaseswithout leukodiapedesis were found in white matter against the background of pronounced swelling with areas of neuropile fragmentation.

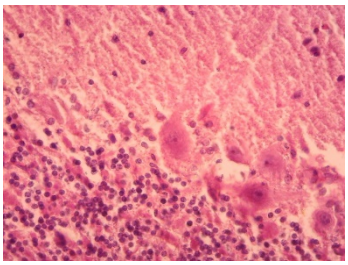

fig. 4 Purkinje cell atrophy with astrocyte loss

In the ganglion layer there is a pronounced swelling of Purkinje cells with a widespread loss of astrocyte spurs (Fig. 4). Cell deposition was observed, areas with sharply swollen offspring. Vacuumization of cytoplasm of separate cells in a number of visual fields was revealed in the granular layer. Focal necroses of individual neurons were noticed.

III. CONCLUSION

In early amputation of the limb a characteristic complex of features is determined: acute neuronal swelling, increase in perivascular expansion of spaces, pericellular expansion of spaces, and hyperoxyphilia of microglia. These morphological features may cause "ischemic" changes in neurons.

The reduction of capillary lumen leads to the expansion of perivascular spaces with the presence of oxyphilic fluid in them, which further promotes the development of so-called perivascular cerebral edema caused by tissue hypoxia.

In the ganglion layer - a pronounced swelling of Purkinje's cells with a widespread loss of astrocytes in the molecular layer of the cortex of the cerebellum rays lobes at the expense of tissue hypoxia, which can be reflected in the form of karyopicnosis and caryorexis nuclei.

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