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МАТЕРІАЛИ ТРЕТЬОЇ ВСЕУКРАЇНСЬКОЇ НАУКОВО-ПРАКТИЧНОЇ КОНФЕРЕНЦІЇ З МІЖНАРОДНОЮ УЧАСТЮ

«ТЕОРІЯ ТА ПРАКТИКА СУЧАСНОЇ МОРФОЛОГІЇ»

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67 years, m:f ratio 1,58:1 and utterly predominant location – anogenital region.

Conclusion. Received data have mostly corresponded to published information previously. Both clinical studies and immunophenotype gave a possibility to make an accurate diagnose.

INTENSIFICATION OF ASTROGLIAL ENDOSOMAL MACHINERY WORK IN SEPSIS-ASSOCIATED ENCEPHALOPATHY

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Astroglial cells are of great importance in the brain defense against infection, restricting the damage to the CNS and regulating brain immune response. It is supposed that in case of sepsis associated encephalopathy (SAE) astrocytes have an enormous contribution to the evolution of pathology and its outcomes. Astrocytes communicate with other brain cells through the releasing of a wide range of molecules, which all are part of the unified “gliocrine system”. One of the secretion pathways from astrocytes is regulated exocytosis via the mechanism of multivesicular bodies (MVBs) and exosomes formation. MVBs are unique organelles in the endocytic pathway which contain vesicles in their lumen (intraluminal vesicles (ILVs)). Endosomal mechanism plays a critical role in the highly dynamic intracellular transport between the plasma membrane, lysosomes and the cellular biosynthetic machine. During SAE it is observed change in neurotransmitter balance in the brain which can be largely due to development of astrogliopathic state. The ultrastructural basis of such phenomenon remains unclear.

The aim of the work: determination of the astroglial endosomal apparatus state in the conditions of SAE.

Material and methods: The cecum ligation and puncture (CLP) model was used to induce abdominal sepsis in rats. Male Wistar rats, 200-250 g, in the amount of 25 were used for this purpose. All rats were randomly divided to 3 groups: 5 sham-operated rats made up the control group, two comparison groups (10 animals each) - with the time of taking the material at 12 (I-st group) and 24 hours (II-nd group) after CLP. Briefly, rats were anesthetized with intraperitoneal injection of Ketamine (80-100 mg/kg) and Xylazine (5-10 mg/kg). After deep anesthesia cecum was exposed through laparotomy aperture, cecum was ligated and perforated by needle distal to ligation place; a small amount of feces were extruded into the abdominal cavity.

After relocating cecum abdominal wall was sutured, using a 4-0 sterile silk suture. A sham surgery was made in control group with the same procedures besides ligation and perforation. Cortex and sub-cortical white matter of the parietal lobe were taken for transmission electron-microscopic study using classical tissue processing method and microscope PEM-100-01 (Selmi, Ukraine).

Results: Analysis of brain tissue samples of CLP-model animals revealed that starting from 12 hours of the experiment and with augmentation up to 24 hours after surgery different kinds of ultrastructural pathology were developed, mainly represented by disintegration of nuclear and cytoplasmic structures in neurones and all types of glial cells. In comparison with the control group in the I-st comparison group the processes and pericarya of astrocytes showed 2 times increase in the number of MVBs which appear as large (0.4-1 μm in diameter) vacuolar structures packed with 2-30 ILVs with diameters of 50 to > 500 nm and at 24 h after operation their number increased up to 4-5 times. Accumulation of MVBs in astrocytes in SAE reflects activated endocytosis and possibly phagocytosis, both likely revealing elevated phagocytic activity of astroglia. In case of SAE astroglial MVBs are highly polymorphic: some of them are densely packed with ILVs whereas others display relatively large vesicle-free zones, which sometimes contain loose electron-dense material or lamellar structures including myelin-like bodies. The MVBs are designed to the sorting, degradation, trans-cellular transport and recycling of various intracellular molecules and are linked to the formation of exosomes involved in cell-to-cell communications which can have its manifestations in abnormal neurotransmitter context and tissue energy imbalance in conditions of SAE. In our study it was revealed substantial increase of the density and redistribution of MVBs into pericapillary astroglial endfeet, especially at 24 h of experiment, which plausibly may reflect gradual activation of adaptive astroglial response to systemic infection and related aggressive factors. It is supposed that such response, in particular, is manifested by elimination and export of cytosolic debris after cellular damage in conditions of SAE.