MORPHOLOGICAL CHANGES IN THE HISTOTOPOGRAPHIC PARTS OF THE BRAIN IN CHILDREN WITH CEREBRAL PALSY

Yoldoshev Yorqinjon
Doctor of clinic "Neuro-star-med"
Fergana medical institute of public health.
https://doi.org/10.5281/zenodo.10617504

Abstract. Traumatic brain injury occurs during fetal and intranatal period as a result of various maternal influences, as well as ischemia and hypoxia in the early postnatal period. Morphological changes in the brain in BTsF are divided into two groups. The first type of changes are brain malformations, manifested by pachygyria, microgyria, underdevelopment of the occiput and cerebellum. The second type of changes is manifested by the appearance of porencephaly, glial reaction, status mormaratus, calcifications, hypo- and aplasia foci of brain lobes in a normally developed brain.

Key words : fetus, baby, brain, injury, anomaly, defect, paralysis, pachygria, microgyria, hypoplasia, aplasia.

Main part. In CP, different levels of pathomorphological changes are detected in the periventricular area of the lateral ventricles of the brain, including dystrophy and death of neurons and glia, migration of neuroblasts to the surface layers of the cerebral cortex, and the presence of different levels of dysontogenetic changes in other areas of the brain. If CP dies at 8-9 months before reaching one year of age, due to long-term inflammation in the brain tissue, along with swelling, hemorrhages, swelling and lymphoplasmic infiltration, gliosis, sclerosis, death of ependymocytes, cystic cavities, small granular calcification occur in the subependymal area. given, the number of small blood vessels in this area is reduced, and the wall of large blood vessels is sclerotized. In severe forms of CP, bilateral hemiplegia, severe spastic diplegias are characterized by the development of sclerosis in blood vessels, in the network of vessels of the ventricles, and in the walls of blood vessels of the meninges . In CP, it is observed that all structural structures of the brain are damaged, more phylogenetically young structures, that is, cortex and basal nuclei, undergo stronger changes (3, Krivitskaya G.N. et al., 2000). The motor analyzer of the brain is the center of activity of the cortical layer, it reproduces motor impulses (4, Kukuev L.A., 1986). Therefore, it is necessary to study the motor analyzer area of the brain in depth. In all CP, strong changes in this area are observed during infancy. In the 4th pole of the brain, it is determined that the cortical layer is crushed. In the III and V layers of this area, it is determined that the cells are diffusely collected, and in the V layer, the nest-like arrangement

of Bets cells is disturbed. The apical dendrites of pyramidal neurons are found to be crooked, and even the pyramids are twisted. Children with BTsF have a significantly reduced number of Bets cells in their brains compared to controls. It was found that the volume density of Bets cell was significantly reduced in hypoxic ischemic encephalopathy and equaled 10.82 on average, and 29.55 in brains of children without brain damage. Therefore, it is determined that the density of Bets cells decreases by 3 times from the normal value in one volume unit when the nervous system is damaged by hypoxia and ischemia.

In almost all cases of BTsF, the cytoarchitectonics of the brain is disturbed to varying degrees, the number of neurons in the III and V layers of the 4th area is reduced, in which the 2 or 3 rows of neurons are disturbed, and neurons in these rows are completely lost. In some cases, it is determined that the heterotopia of neurons has appeared in the lower layers of the cortex, in the molecular layer of the cortex. In the visual field of the cerebral cortex, cytoarchitectonic batomom is found to be disturbed (9). It is determined that these changes occur to different degrees depending on the time of their appearance. In the premature development of microcephaly and secondary and tertiary egates, the cerebellum is reduced, nerve cells undergo dystrophic changes, in some cases calcification occurs, soft brain lesions and focal sclerosing of the vascular network, the growth of connective tissue in the walls of blood vessels, the appearance of petrifications, if the children are young, the fetus indicates that it began to develop during In addition to these changes in the brain, it is observed that during the fetal period of the child, birth defects have developed in the internal organs, often in the heart. Of these, there is a defect in the space between the heart ventricles, aplasia of the kidney, and the development of cytomegaly.

Microgyria in the brain, underdevelopment of brain surface egates, disruption of the cytoarchitectonics of neurons in the motor area of the brain, decrease in the number of pyramidal neurons, non-firing of Bets cells, the appearance of strong dystrophic changes, hemocytulation disorders, the development of focal proliferative inflammation in the wall of the lateral ventricles indicate damage to the brain in the congenital and perinatal period. . These morphological changes are the main pathomorphological features of BTsF. Histological examination of the brain tissue reveals strong pathomorphological changes in the neurons of the cortex, subcortical structures, nuclei of cranial nerves, which are brain motor analyzers.

In the severe form of the disease, calcification, which is an irreversible change in the neurons of the brain, is manifested by the appearance of "shadow cells". It was found that developed dystrophic changes in nerve cells and their fibers accompanied the development of sclerosis in the walls of cerebral blood vessels, ventricular vascular network and soft membranes. When studying the ratio of neurons and glial cells in the nuclei of the cerebrospinal nerves, it was found that the density of nerve cells is mainly reduced (3). The density of glial cells was determined in the area of the corticospinal tract of the brain nerves. It was found that strong changes in the nuclei of motor analyzers caused complex changes in the cerebral cortex, striopallidal system, visual cortex, cerebellum. In these patients, it is observed that various levels of changes have developed in the nerve fibers, and they are fragmented and disintegrated of the myelin sheath, deformed cylinders, varicose expansion, fragmentation and death. Strong dystrophic changes of the axis cylinders are often found to have developed in its terminal part. It is observed that the terminal axons of motor neurons of the corticospinal tract have undergone a strong change.

Thus, dystrophic changes develop in neurons as a result of destructive changes in terminal axons. Transneuronal degeneration develops in cranial nerves and motor neurons, which leads to impaired nerve function. In severe cases of BTsF, peripheral paralysis is added to the clinical symptoms at the age of 3-5 years, that is, hypertonia or hypotonia in the muscles of the shoulder, lumbar and pelvic area. According to the clinical and morphological analysis of most of the dead patients, it was confirmed that the disease was severe, and strong pathomorphological changes developed in the tissue structures of the brain. At the same time, it is determined that various levels of adaptation-filling and reparative changes have been developed in the brain tissue of mainly young patients. It is found that immature neurons are preserved in the brains of patients who died before the age of one year .

Conclusions

Morphological changes in the brain in BTsF are divided into two groups. The first type of changes are brain malformations, manifested by pachygyria, microgyria, underdevelopment of the occiput and cerebellum. The second type of changes is manifested by the appearance of porencephaly, glial reaction, status mormaratus, calcifications, hypo- and aplasia foci of brain lobes in a normally developed brain.

Literature:

1. Zokirov, M. (2023, June). Features of cognitive impairment in patients with HIV encephalopathy. In Academic International Conference on Multi-Disciplinary Studies and Education (Vol. 1, No. 9, pp. 34-36).
2. Zokirov M.M., & Madjidova Y.N., . (2020). Correction Of Cognitive Disorder In Patients With HIV - Associated Encephalopathy. The American Journal of Medical Sciences and Pharmaceutical Research, 2(07), 117–122. https://doi.org/10.37547/TAJMSPR/Volume02Issue07-15
3. Zokiriv, M. (2021). Correction of cognitive impairments in patients with HIV-associated encephalopathy. J. Theor. Appl. Sci, 7, 62-66.
4. Zokirov Muzaffar, & Muhammadjonov Oqilbek. (2023). Late clinical and neuroimaging manifestations of post-traumatic epilepsy and optimization of its treatment. Novateur Publications, 7, 1–108. Retrieved from http://novateurpublication.org/index.php/np/article/view/114
5. Muzaffar, Z. (2023). Anxiety and Depression in Patients with HIV Encephalopathy. Eurasian Medical Research Periodical, 21, 95-98.
6. Oqilbek, M., & Muzaffar, Z. (2023). Parameters of Apoptosis and Prevention of Neuro-Like Conditions in Patients with Type II Diabetes Mellitus. Eurasian Medical Research Periodical, 21, 99-102.
7. Muzaffar, Z., & Zarifa, Z. (2023). Yosh bemorlarda gemorragik insultning klinik holatlari. Psixologiya va sotsiologiya ilmiy jurnali, 1(1), 29-31.
8. Muhammadjonov, O., & Zokirov, M. Anemiya kasalligida bemorlarning surunkali buyrak etishmovchiligida epokrin preparatini qo'llash. Студенческий Вестник