МИКРОСКОПИЧЕСКИЕ ИЗМЕНЕНИЯ В ГОЛОВНОМ МОЗГЕ ДЕТЕЙ, УМЕРШИХ ОТ ЦЕРЕБРАЛЬНОГО ПАРАЛИЧА

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МИКРОСКОПИЧЕСКИЕ ИЗМЕНЕНИЯ В ГОЛОВНОМ МОЗГЕ ДЕТЕЙ, УМЕРШИХ ОТ ЦЕРЕБРАЛЬНОГО ПАРАЛИЧА.ЖКМП.-2025.-Т.2.-№2.-С

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Аннотация: При детском церебральном параличе (ДЦП) морфологические признаки изменений в головном мозге зависят от возраста, пола, конституционного строения, продолжительности воздействия факторов и тяжести заболевания и проявляются в основном в зависимости от плотности клеток в слое головного мозга и снижения тяжести лад. В большинстве случаев головной мозг проявляется множественными стромами с фибриллярной структурой и множественными глиальными клетками. Ангиоархитектоника сосудов и капилляров, содержащих серое вещество, также проявляется в измененной, менее разветвленной капиллярной сети. Наиболее распространенными областями астроцитов являются эти области.

Ключевые слова: *детский церебральный паралич, морфология, головной мозг, астроциты, глиоз, синдром припадка.*

BOLALAR SEREBRAL FALAJIDA TUTQANOQ SINDROMI BILAN VAFOT ETGAN BOLALARNING BOSH MIYASIDAGI MIKROSKOPIK O‘ZGARISHLAR

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Annotatsiya: Bolalar serebral falajida (BSF) da bosh miyadagi o‘zgarishlarning morfologik jixatlari bemorning yoshiga, jinsiga, konstitutsional tuzilishiga, ta’sirlovchi omillarning davomiyligiga va kasallikning og‘irligiga bog‘liq bo‘lib, asosan, bosh miya po‘stloq qavatidagi xujayralarning zichligi va xajmiy jixatlariga bog‘liq ravishda kamayishi bilan namoyon bo‘ladi. Asosan, bosh miya po‘stloq qavatida astrotsitlarning kam sonli bo‘lgan, ko‘p xollarda fibrillyar tuzilishli xususiyatlarining ko‘pligi va glial xujayralarning ko‘p sonli bo‘ilshi bilan namoyon bo‘ladi. Kulrang modda tarkibidagi qon tomir va kapillyarlarning angioarxitektonikasi xam o‘zgargan, kam tarmoqlangan kapillyar tarmoqlarining to‘laqonli ko‘rinishi namoyon bo‘ladi. Astrotsitlarni eng ko‘p joylashgan soxalari bu chakka va ensa soxasi xisoblanadi.

Kalit so‘zlar: *bolalar serebral falaji, morfologiya, bosh miya, astrotsitlar, glioz, tutqanoq sindromi.*

MICROSCOPIC CHANGES IN THE BRAIN OF CHILDREN WHO DIED OF CEREBRAL PALSY

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Abstract: In infantile cerebral palsy (ICP), the morphological signs of changes in the brain depend on age, sex, constitutional structure, duration of exposure to factors and severity of the disease and appear mainly depending on the density of cells in the brain layer and the decrease in the severity of frets. In most cases, the brain presents with multiple stomas with fibrillary structure and multiple glial cells. Angioarchitectonics of vessels and capillaries containing gray matter also manifests in an altered, less branched capillary network. The most common areas of astrocytes are these areas.

Keywords: *infantile cerebral palsy, morphology, brain, astrocytes, gliosis, seizure syndrome.*

Materials and methods: At the Republican Center for Pathological Anatomy during autopsy of corpses of 5-9 years of various ages who died from infantile cerebral palsy (ICP), brain fragments measuring 1x1x1 cm from the tissue are cut off from symmetrical sections of the temporal lobe and anterior hypothalamus of both hemispheres [3,4,6]. The samples were dried, filled with alcohol of increased concentration, alcohol-chloroform, filled with kerosene blocks and glued to oily cubes. In the pre-block projection there are consecutive cuts with thickness of 8-10 mkm [1]. Hematoxylline is isolated in eosin oil and the nuclear structures of the hypothalamic region of the brain are morphologically determined: the thickness, length (height) of the cortical layers and the width of neurons in the layers, the density of neurons and neuroglia $1mm^{2}$.

Discussions and results: Microscopic changes in the brains of children who died from infantile cerebral palsy (ICP). The study of brain tissues revealed cases of microhyria, macrohyria, dysgenesis and even agenesis of nuclear structures. In addition, expansion of the ventricles and subarachnoid cavities is observed. In one of the cases of microcephaly associated [2,7,11], with seizure syndrome, extensive hemorrhage in the temporal region of the left hemisphere (hemorrhagic infarction). Microscopic examination revealed deformations caused by brain damage (fibrous changes), inaccurately formed thin uneven folds (microhyria, macrohyria), torn cystic structures in the depths of the brain, pathological processes of cerebral vessels that occurred during embryonic development [5,8,9].

Changes in gray matter (cortex) in the brain: In the superficial layers, nerve cells are absent or atrophic, in a degenerative form around atrophic and degenerative neurons, the number of glial cells increases (proliferation of glia), the walls of blood vessels and their sclerotic changes thicken significantly, the meninges thicken significantly, the number of rare fiber structures increases, tumor and softening of the axillary tissue (spongy changes), glial scar formation [11-13]. With asymmetry and deformation of the macroscopic folds and depths of the brain caused by different degrees of continuity, dilation of blood vessels in the brain, full appearance, thickening of the walls, reproduction of gliosis loci, causing cerebral ischemia and atrophy of nerve cells of the present Position [10]. In the upper layer of the brain, where nerve cells are located unstable and irregularly, large, nucleus-free “zones” are formed, in the intervals between which irregularly multiplying loci of protoplasmic fibrillary structures are revealed [14]. It turned out that neurons of various sizes and shapes changed, and most of the neurons in the field of view were elongated and reduced in size. In some morphofunctionally active neurons, the tigroid substance (Nissl) is very dense and dark in color, manifests itself as a clinical morphological uptake syndrome due to the accumulation of a large number of hyperimpulsions and pathological impulses in practice, the presence of tigroid substances in the cytoplasm of neurons in a dense basophilic form is characteristic of those who died from epileptic status [14,15]. It is characterized by the fact that nerve cells form clusters in small folds formed between the depths of the brain, but with smaller sizes. There is also a cell-free zone on the surface, but it is relatively narrow [16-18]. In the deep layers of the brain, destructive changes in nerve and glial cells are observed.

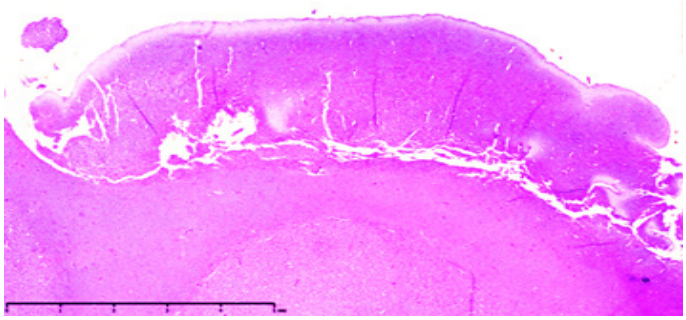

Figure 1. A 4-year-old boy. In infantile cerebral palsy with bilateral hemiplegic form and seizure syndrome. Brain atrophy. Significant thickening of the soft dura mater. Expansion of the cell-free layer of the brain. Stain: H&E. Size: 10×10.

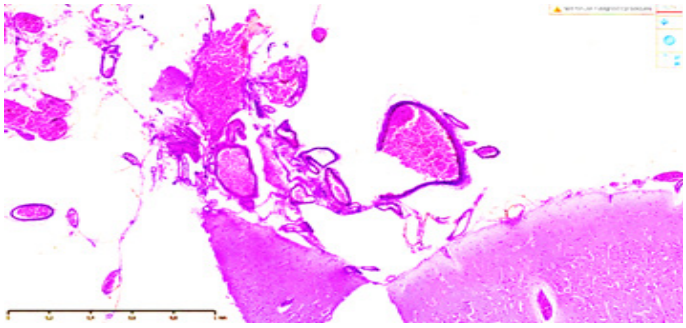

Figure 2. Autopsy of a 4-year-old patient - brain tissue. Various forms of cerebral vascular malformation, deformed vessel walls of varying thickness, some areas thickened and aneurysms. Most of the vessels look “good”.Detection of gliosis in the cerebral stratum corneum and irregular arrangement of nerve cells. Stain: H&E. Size: 10×20.

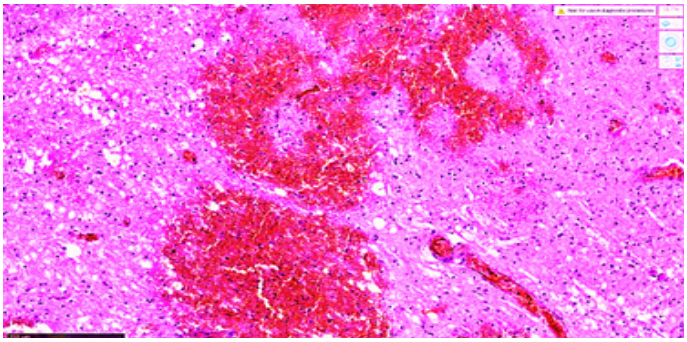

Figure 3. 5-year-old patient. Severe cerebral hemorrhage. Gliosis around the hemorrhage (enlargement of glial cells). Stain: H&E. Size: 10×20.

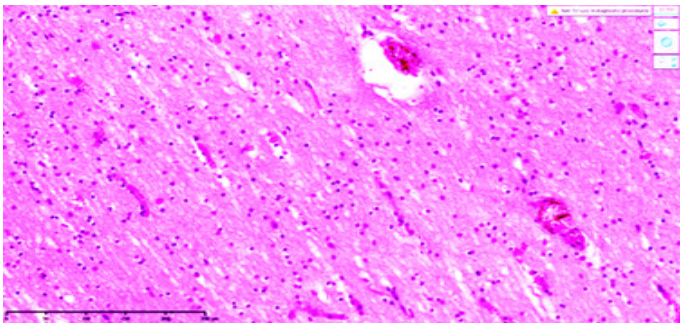

Figure 4. 6-year-old patient. With circulatory impairment and gliosis foci in brain sections (1), anemia persists in the veins. Intermediate tumors. Stain: H&E. Size: 10×20.

The microscopic picture of the hemorrhage shows "limb resorption, hemoglobinogenesis pigment formation, and tonic surrounding brain tissue," consistent with a recent, acute, clinically convulsive syndrome and possibly the immediate cause of death [18,20]. With the breakdown of red blood cells and nerve tissue in the center of the hematoma, a small gliosis is observed, which indicates the weakness of the regenerative activity of glial cells and atrophic changes. One of the main features is that hypoxia of at least 2-3 microglial cells occurs around astrocytes in the area of the brain staple, and the multiplication of glial cells and gliosis of mainly protoplasmic fibrillary structures means that it was common [5]. As a result, microhyria, agarium deformities, and asymmetric phenomena are found in the cerebral hemispheres.

In the brain tissues around the hemorrhage, a small activation of nerve cells is found, that is, hypertrophy and hypertrophy of glial cells (Fig. 3). In a perivascular tumor, there is a narrowing of vascular illumination. Thus, with infantile cerebral palsy (ICP) and morphological manifestation of convulsive syndrome with a decrease in brain mass in the form of microsephaly, atrophy and deformation of the hemispheres, microhyria, macrohyria, agenesis of individual wolves and nuclei, ventricular dilation, and large hemorrhages in the deep layers of this Regulation. Microscopic examination revealed a lag in the development and differentiation of nerve cells, the appearance of cell-free fields, irregular arrangement of nerve cells, and an indefinite content of tigroid substances in nerve cells. Under microscopic examination, the number of nerve cells in convulsive syndrome is limited; some of them are not developed, and some have retained signs of neuroblasts. Other nerve cells have an intermediate appearance between the neuroblast and the resulting nerve cell. Processes in cells are small and brief. The number of nerve cells in layers II and III decreased; they are located unevenly. The distribution of pyramidal cells shows anomalies of layers, heterotopia. In all layers, they are located in uneven layers. Microscopic examination of some areas of the brain with cerebral palsy and seizure syndrome revealed distortions and deformities in the ribbons of the hemispheres [18].

Thickened areas of the cerebrospinal membrane, especially from the atrophic folds, are determined by the proliferation of connective tissue cells and thickening of the vascular walls. With less damage, layers of the brain are released, although the outer acellular layer is enlarged, and the pyramidal cell layers are thin and contain small cells. The outer non-cellular layer consists of hypertrophied glial cells of the soft brain. The cellular layers of the cortex lack clear boundaries; in all regions, nerve cells vary in size and shape and are arranged irregularly [19]. Various authorized centers and gliosis centers are formed among nerve cells (Fig. 4). In a histochemical study, Nissl showed that nerve cells in a state of convulsive syndrome are located irregularly, in various sizes and shapes, most of which are compressed and deformed, and have an uneven content of tiger matter (Fig.5).

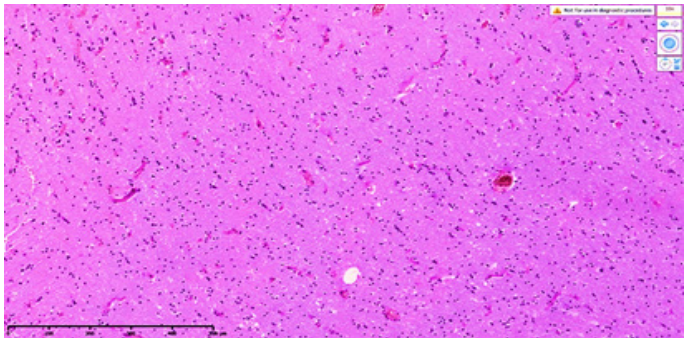

Figure 5. A 6.4-year-old child with cerebral palsy and seizure syndrome, and micropolygyria. With the developed asymmetry in the large hemispheres, an uneven arrangement of cells in the affected areas, and the formation of gliosis foci are revealed. Stain: H&E. Size: 10×10.

Nearby vessels are slightly dilated, together with wall thickening and perivascular swelling. Edema and nerve cell irregularities in remote areas of the hematoma are evident. In children with cerebral palsy, if they have a history of intrauterine infantile brain damage as a result of intrauterine and external reactions (infections, vascular diseases, endocrine diseases), microgyria, agyria (lissenkephaly), pachygyria, amaurotic idiocy, on the side of the cavities - hydrocephalus, on the white matter - diffusion, placed gliosis and porecephaly [20,21].

Conclusion: Microscopic changes associated with the seizure syndrome in cerebral palsy constitute the main morphologic picture of neurologic disorders of the pediatric brain. The major changes are characterized by pathology in neurons and glial structures, abnormalities in cortical architecture, and illicit microcirculatory events. These results have important implications in understanding the clinical picture of infantile cerebral palsy, improving diagnostic methods, and pathogenetic treatment.

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