LEUCOENCEPHALITIS

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Leukoencephalitis is one of the clinical forms of demyelinating disease. With leukoencephalitis, the gray matter of the brain is usually affected, so the more correct term is “panencephalitis”. It has a steadily progressive course with a nonspecific and polymorphic clinical picture, which may include mental disorders, pyramidal and extrapyramidal syndromes, cognitive deficits, damage to the cranial nerves, and episyndrome.

The etiology of leukoencephalitis has not yet been clarified. It is assumed to be viral in nature, but attempts to isolate the virus have so far been unsuccessful.

In the process of elucidating the etiology of leukoencephalitis, an opinion arose about the involvement of this disease in slow infections associated with the latent long-term survival of the virus in the body (herpes simplex virus, measles, etc.). Its activation occurs under the influence of a number of factors. When they enter the nervous system, activated slow viruses cause the development of an acute or chronically progressive process.

From the history of the discovery of the disease.

The first description of leukoencephalitis belongs to Schilder (1912). He considered this disease as an independent nosological entity and gave it the name “diffuse periaxial sclerosis.” It later became known as Schilder's disease.

In the 1930s, a number of other forms of leukoencephalitis were described. Van Bogaert considered this disease to be an independent form of encephalitis and called it subacute sclerosing encephalitis.

Dawson, and then Pette and Doering described another group of similar diseases called nodular panencephalitis.

Uzunov, Tozhinov and Georgiev described encephalitis similar in clinical picture to subacute sclerosing leukoencephalitis.

G.B.Abramovich and Ageeva, E.F.Davidenkova and Pavlovich outlined in detail the clinical features of subacute sclerosing leukoencephalitis.

The pathomorphological changes are based on the process of demyelination of the white matter of the cerebral hemispheres with less significant damage to the axial cylinders. It should be remembered that according to the nature of myelin damage, myelinoclastic and leukodystrophic types of demyelinating

diseases are distinguished. In the first case, myelin is destroyed, in the second, myelin metabolism is disrupted due to a hereditary enzymatic defect.

Leukoencephalitis is of the myelinoclastic type. Diffuse or focal demyelination in all parts of the brain, atrophy of the gyri, and widening of the sulci are typical. Histologically, diffuse inflammatory and degenerative changes in the white and gray matter of the brain, perivascular infiltrates, degeneration of neurons with neuronophagic nodules and inclusions in the nuclei and cytoplasm are noted. There is intense proliferation of astrocytes and a proliferative reaction of glia. Gliosis can be small-focal or large-focal. Diffuse gliosis leads to thickening of the brain substance. Neurons of the cerebral cortex contain spherical inclusions with a diameter of 30-40 microns or smaller, tubular in shape. In acute hemorrhagic leukoencephalitis, cerebral edema is found, on a section of the hemispheres - foci of a soft pink-gray color with numerous pinpoint hemorrhages, and histologically - fibrinoid necrosis of the walls of small vessels with ring-shaped hemorrhagic zones, perivascular demyelination with axonal destruction.

There are several forms of the disease:

- subacute sclerosing panencephalitis of Van Bogart;
- Schilder's periaxial leukoencephalitis;
- acute hemorrhagic leukoencephalitis.

It is assumed that Van Bogart panencephalitis is caused by the measles virus, which can persist in brain neurons for a long time and, under certain conditions, become activated. Some researchers consider Schilder's leukoencephalitis as a variant of multiple sclerosis in children. Acute hemorrhagic leukoencephalitis often occurs after preventive vaccinations. The pathogenesis is not clear. A hyperergic autoimmune process is assumed, in which viruses play the role of a trigger.

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