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DAMAGE TO THE NERVOUS SYSTEM IN HIV INFECTION

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HIV infection is a disease caused by a retrovirus that affects cells of the immune, nervous and other systems of the human body [1, 2]. Damage to the nervous system, primarily the brain, is one of the most important problems associated with HIV infection. Neurological damage in HIV infection is observed in 50-80% of patients and is clinically expressed in more than 10% of them [3, 4]. Damage to the nervous system is primary in 7.2% of patients [5, 6]. NeuroAIDS is the leading clinical syndrome in 30% of patients [6]. There are several hypotheses about how HIV penetrates the CNS: penetration of HIV through the blood-brain barrier with infected lymphocytes and microphages; penetration of HIV into the CNS through nerve fibers; penetration through gaps between the endothelial cells of capillaries. Causal factors of neurological disorders in HIV infection: direct action of HIV, mediated damage to brain tissue by cytokines, impact of pathogens of secondary infections, neoplasms, vascular complications. Psychogenic and iatrogenic factors can also acquire pathogenetic significance [4, 5].

In 70-90% of patients who died from AIDS, changes in brain tissue were found [4]. Pathomorphological changes are represented by inflammatory perivascular infiltration, proliferation of vascular wall cells, oligodendro- and microglia , reactive cliosis , degenerative changes in white matter, focal demyelination [5, 6].

The topical classification identifies the following variants of neurological manifestation of HIV infection:

- brain and meninges: HIV meningoencephalitis , opportunistic infections of the central nervous system, neoplastic processes, acute cerebrovascular accidents , road traffic syndrome;

- spinal cord: HIV-associated vacuolar myelopathy , acute myelopathy in opportunistic infections;

- peripheral nervous system: distal symmetric polyneuropathy, polyneuropathy in opportunistic infections, facial nerve neuropathy, neural amyotrophy , multiple mononeuritis, lumbosacral polyradiculopathy , demyelinating polyradiculoneuropathy [6].

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Aseptic meningitis occurs during the seroconversion period , both in acute and chronic forms. Subclinical meningitis is possible [4]. There are no specific clinical signs. Mononuclear pleocytosis up to 20 or more cells. HIV can be detected in the cerebrospinal fluid. The course is favorable, but relapses are possible [4, 5]. Acute encephalitis most often occurs in the first three months of the disease, clinically characterized by fever, general malaise, mood changes, epileptic seizures, and changes in the level of consciousness [3, 4]. Full restoration of functions occurs within 2-4 weeks.

Acute meningoencephalitis occurs in 5-10% of cases, often accompanied by damage to the V, VII and VIII pairs of cranial nerves [4]. Subacute encephalitis (AIDS-dementia complex, AIDS-DC) is observed in 6-21% of adult patients [4]. In most cases, AIDS-DC develops against the background of profound immunodeficiency. However, it may be the first clinical manifestation of HIV infection.

AIDS-DC is characterized by a triad: cognitive impairment, motor disorders, and altered behavior [5]. Dementia is predominantly of the frontal-subcortical type [4]. Significant cognitive impairment, difficulty concentrating, and memory impairment are noted [3, 5]. Sleep disorders, akinetic-rigid syndrome, tremor, and cerebellar ataxia are common. Symptoms of oral automatism and oculomotor disorders may occur [4, 5]. The neurological status also reveals muscle hypotonia combined with a high reflex background [6]. In the late stages of AIDS, dementia, paresis, and paralysis develop, and generalized seizures are possible. In the terminal stage, patients approach a vegetative state. The course of the disease progresses over several months and even years, with long-term remission possible [4]. The most severe CNS lesions are determined when the content of CD4 lymphocytes in the peripheral blood is less than 0.2×10^{9}/l and the viral load is more than 10,000 copies/ml. In the natural course of the disease, the terminal phase occurs within 4-8 years in more than 80% of cases [5].

Among the laboratory parameters, the most characteristic is considered to be a high content of protein in the CSF (up to 1.0 g/l). Lymphocytic cytosis is usually small, about 200 cells / ml, a decrease in glucose concentration is possible, but changes may also be absent [4-6]. CT and MRI of the brain reveal enlargement of the cerebral ventricles and diffuse depletion of white matter, less often - changes in the thalamus and basal ganglia [3, 4]. In children, calcification of the basal ganglia or their atrophy is noted. During puncture, small foci (less than 1 cm in diameter) are detected in the biopsy [3]. Bilateral diffuse slowing of the rhythm is detected on EEG [5, 6].

Lesions of the peripheral nervous system are observed in more than 50% of patients [4], are noted in all patients with CD4 lymphocyte content of 0.2-0.29 ç 109/l and below. Peripheral polyneuropathy is symmetrical. Its occurrence is associated with the direct effect of HIV on the peripheral nervous system. The disease begins with paresthesia, then weakness of the distal limbs and muscle atrophy increase [6]. The

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course is slowly progressive. Electroneuromyography reveals both myelopathy and axonal type of damage.

Acute polyradiculoneuropathy (Guillain-Barré syndrome) often develops during the seroconversion period [4]. Morphologically, perivascular inflammatory infiltrates and demyelination of nerve trunks are determined [5]. Increased protein content, lymphocytic pleocytosis up to 50 cells /ml [4]. Multiple mononeuropathy with damage to the cranial and spinal nerves manifests itself acutely or subacutely, often ending in spontaneous remission. It can manifest itself at an early stage of HIV infection, the late stage is characterized by a rapidly progressive course [4]. There are no intoxication symptoms, the pain syndrome is unstable [5].

It is known that the incidence of strokes in young people with HIV infection is 40 times higher than in the general population. Among the many causes are damage to the vascular endothelium, thrombocytopenia, heart damage, rupture of mycotic aneurysms and hemorrhage into a tumor [4, 7]. With progressive immunodeficiency, susceptibility to bacterial, fungal, viral and parasitic infections that cause brain damage increases [4, 5].

Cytomegalovirus infection (CMV) is registered in 20-40% of AIDS patients [3, 4]. Encephalitis has a subacute course with a gradual increase in clinical symptoms. Frequent mood swings, lack of criticality towards one's condition, sleep and memory disorders, and periodic headaches are observed. 3-4 weeks before the death of patients, headaches intensify, fever becomes more persistent, memory deteriorates significantly, drowsiness and adynamia increase, and personality disintegration is possible. Focal symptoms increase, visual and auditory hallucinations appear. Meningoencephalitis proceeds more aggressively - with a rapid impairment of consciousness and coma [3].

Cytomegalovirus spinal cord lesions are often diffuse. They manifest as pain syndrome, lower paraparesis , pelvic dysfunction, and optic neuritis [3, 4]. The development of myelitis is an unfavorable prognostic sign: as a rule, patients die within 1-3 months. CT of the brain may reveal subependymal changes with ventriculitis . Changes in the CSF are characterized by a slight increase in protein levels (0.6-1 g/l), double-digit cytosis , lymphocytic composition, and sometimes cytomegalovirus can be isolated from it [3].

A common opportunistic disease is tuberculous meningitis. The disease usually has a severe course; against the background of severe immunodeficiency, the typical picture of the disease may be distorted. The prognosis is usually unfavorable [3, 7].

Toxoplasmosis is widespread in HIV infection. The main and most common form of the disease is cerebral toxoplasmosis, which develops in AIDS patients in different countries in 3-40% of cases and is successfully (in comparison with other infectious lesions of the central nervous system) treated [3, 8]. Acute or subacute onset is typical, the clinical picture is dominated by symptoms of focal encephalitis, indicating damage

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to the hemispheres, cerebellum or brainstem [3, 6]. Focal or generalized seizures , headache, fever, mental disorders are noted. Meningeal symptoms are rare [3]. In 20-30% of patients, the composition of the CSF is normal, an increase in protein content to 1-2 g / l and higher, two- and three-digit lymphocytic pleocytosis [3, 4]. Determination of antigen in cerebrospinal fluid is used. Serological methods are usually uninformative [6]. CT can detect compactions in the cortex, thalamus, and basal ganglia. The optimal visualization method in these cases is MRI [3, 6].

CNS lesion caused by Varicella virus zoster , most often has the character of encephalitis. The main symptoms are headache, nausea, vomiting, ataxia, tremor, and impaired consciousness. CNS damage can have the character of cerebral vasculitis , multiple cranial nerve lesions, and myelopathy .

As a result of the use of HAART (highly active antiretroviral therapy), a relative improvement in the neurological status and mental development of young children is possible. Improvement of psychomotor development is one of the indicators for assessing the effectiveness of HAART (along with an assessment of physical development, immunological and virological indicators).

It is necessary to emphasize the complexity of the problem of diagnosis and treatment of HIV infection in general and its neurological manifestations in particular. In order to improve the quality of life, it is necessary to further search for methods of early diagnosis and prevention of HIV infection in children. An important aspect of rehabilitation of such patients is not only drug therapy, but also psychological and social assistance [4].

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