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NEUROLOGICAL COMPLICATIONS OF AIDS

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Summary. In the lecture, data were given about neurological complications In the syndrome of full immunodeficiency (AIDS), which occur In 50-60% of patients. The most widespread are encephalitis, meningitis, meningoencephalitis, polyneuropathy, myelopathy, myopathy. Ethiopathogenetic mechanisms of lhese complications, clinic, treatment, prognosis are being developed.

Key words: AIDS; damage Io the nervous system; jubilation; forecast

According to the World Health Organization, about 45 million people on the planet are infected with the human immunodeficiency virus (HIV). The average rate of infection in Uzbekistan is 40 cases per 100 thousand population. One of the elements of the human body affected by HIV is the nervous system. Neurological complications of HIV infection can be caused by both retroviruses and opportunistic infections, tumors, cerebrovascular diseases, and the toxic effects of antiretroviral drugs.

It is known that the direct damage to the body consists in the infection and destruction of cells of the nervous system, which have the CD4 receptor. These include astrocytes, oligodendrocytes, microglia, monocytes, fibroblast-like cells of the brain, cells of the endothelium of blood vessels, and neurons. In addition to the penetration of HIV into the cell, membrane lysis by the gp120 protein occurs. A violation of mediator exchange is observed, and a deficiency of gamma-aminobutyric acid and glycine leads to the development of epileptic seizures, a decrease in the level of serotonin (antiserotonin ataxia), a violation of vasopressin (memory impairment). Damage to endothelial cells of vascular plexuses contributes to the development of inflammation of mesenchymal elements of nervous tissue and secondary demyelination. Depression of cellular immunity causes the development of opportunistic infections and neoplastic processes in patients. The virus enters the brain through the perineural spaces, as well as from the cells of the immune system into the nervous system ("Trojan horse" mechanism). There are variants of genetically determined HIV damage, occurrence of primary vasculitis and vasculopathies, development of hemorrhagic complications, violation of blood rheology and hypercoagulation. Against the background of HIV-associated vasculitis, multifocal lesions of the brain often develop, which indicates the occurrence of the meningovascular form of neuro-AIDS. In 40% of HIV-infected patients, changes in the cerebrospinal fluid (CSF) are observed, which are specific (lymphocytic pleocytosis — 5–50 cells/mm 3), an increased amount of protein (500–1000 mg/l), a

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normal concentration is observed glucose Anti-HIV is detected in CMP in high titers. Neurological complications in acquired immunodeficiency syndrome (AIDS) occur in 50–60% of patients, and in 5–10% of cases, these are the first symptoms of the disease. Neurological complications at one level or another are diagnosed at autopsy in 90–100% of cases. Involvement of the nervous system in the pathological process can be the result of HIV or an opportunistic disease.

The development of acute meningitis, meningoencephalitis, or polyneuritis may indicate seroconversion — the appearance of specific antibodies to HIV in the patient's blood serum, but the results of standard HIV testing methods may become positive only 3–6 months after the onset of symptoms. Patients with the specified syndromes and suspicion of HIV should undergo more sensitive virus- or antigen-specific tests, and if possible, repeat standard testing after 3–6 months.

The history of the disease is important for diagnosis, and encephalitis is detected in 15-20% of AIDS patients in the early stages, and this may be the first sign of the disease. HIV infection progresses rapidly, the average life span from the first symptoms does not exceed 6 months. True, today the clinical picture does not correspond to the degree of pathological changes found during the autopsy of the dead.

Treatment

Zidovudine (AZT), which inhibits HIV reverse transcriptase, is recommended. This is the only antiretroviral drug whose effectiveness has been proven today. The drug often restores impaired cognitive functions. The higher the dose, the better the therapeutic effect. It is recommended to take 1200–2000 mg/day, dividing the dose into 5–6 doses, which contributes to better tolerability. A complication can be a decrease in bone marrow hematopoiesis. For prevention, it is recommended to simultaneously use erythropoietin until the level of hemoglobin exceeds 100 g/l and neutrophilia reaches 1000/mm 3. Otherwise, blood transfusion is indicated. In addition, zidovudine can cause headache, nausea, vomiting and general weakness. To combat HIV strains resistant to AZT, dideoxynucleosides (DDI, DDC, DuT) are used, which, unfortunately, are ineffective in the treatment of HIV encephalitis. Combined treatment is more effective. Complications of DDI and other similar drugs are peripheral neuropathy, pancreatitis, toxic damage to the liver and gastrointestinal tract. Today, protease inhibitors are being developed to treat HIV complications. With apathy and autism, it is recommended to prescribe methylphenidase in a dose of 5–10 mg 2–3 times a day to AIDS patients. The side effects of this drug include: toxic liver damage, gastrointestinal disorders, delirium, convulsions, anxiety and sleep disturbances. In case of depression, tricyclic antidepressants (amitriptyline at a dose of 25 mg per night) are prescribed. Every week, the dose of the drug is increased by 25 mg (with satisfactory tolerability) until the clinical effect is achieved. Serotonin reuptake inhibitors, such as fluoxetine, are also prescribed at an initial dose of 10–20 mg/day

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with a weekly dose increase of 10 mg. Disadvantages of such treatment include urinary retention, constipation, dry mouth, heart conduction disturbances (blockages), arterial hypotension, toxic delirium, convulsions, tremors, paresthesias, increased appetite, and weight gain. Side effects of fluoxetine: chills, fever, nausea, headache, angina, arthralgia, increase or decrease in blood pressure.

HIV patients may experience convulsions that require anticonvulsant therapy (mainly phenobarbital). Patients should be constantly cared for, as they lose the possibility of basic self-care and eating. It is better to place them in hospices where special assistance programs have been developed.

The prognosis is poor, but treatment with AZT prolongs the life expectancy of such patients.

HIV myelopathy

HIV myelopathy occurs in 40% of patients (according to autopsy data). Myelopathy is associated with damage to the posterior and lateral cords of the thoracic spinal cord. The specific causative agent of myelopathy has not been found, because in the acute stage of HIV infection, the causative agent is not located in the spinal cord. The clinic is manifested by spastic paraparesis and sensitive ataxia. The disease appears at the stage of deep immunodeficiency and progresses gradually over several months. In patients, the function of the pelvic organs is often disturbed (more urination) and there is a deep sensitivity - muscle-articular and vibration, which is a manifestation of damage to the posterior cords of the spinal cord. Up to 60% of patients with myelopathy also suffer from encephalopathy. HIV myelopathy should be differentiated from vitamin B12 deficiency and syphilis.

MRI may be normal or an atrophic process may be observed. There is currently no specific treatment. To reduce spasticity, baclofen is prescribed in a dose of 5–20 mg 3–4 times a day, in case of urination disorders, oxybutynin is prescribed in a dose of 2.5–5 mg 3–4 times a day. Walking — on crutches or with a walker. Baclofen should not be abruptly discontinued (especially at high doses), as seizures may occur. Oxybutynin can cause anticholinesterase effects — decreased intestinal peristalsis, dry mouth, abdominal pain, nausea.

The prognosis is unfavorable, although some patients have a relatively long life expectancy. Distal sensory HIV neuropathy Distal sensory HIV neuropathy is the most common type of neuropathy associated with HIV infection. It occurs in 1/3 of patients, and in some cases in almost 100% of those who die from AIDS. At autopsy, progressive abnormal degeneration with signs of inflammation of the endoneurium and epineurium is observed.

In the clinical picture, there is a symmetrical disturbance of sensitivity in the distal parts of the limbs, starting from the feet with gradual progression. Patients complain of severe burning and shooting pain. Sensitivity in the feet is impaired, paresthesias are

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noted in the upper and lower limbs. Violated pain and deep muscle sensitivity, in particular vibration, loss of Achilles reflexes, sensitive ataxia. There is weakness in the muscles of the distal parts of the limbs.

It should be differentiated from alcoholic neuropathy, vitamin B12 deficiency, diabetic neuropathy. EMT indicates a symmetrical degenerative-reinnervation process. Treatment consists, first of all, in the fight against immunodeficiency virus. To reduce neuropathic pain, anticonvulsant therapy and tricyclic antidepressants (carbamazepine 200 mg 3–4 r/day or diphenylhydantoin 100 mg 3–4 r/day) are recommended. You can use gabapentin in a dose of 300–600 mg 3 times a day. Mexiletine is used at 150–300 mg 2 times a day. It is prescribed after withdrawal of anticonvulsant drugs. With long-term use of diphenylhydantoin, osteomalacia and neuropathy can develop, and maxiletine can cause damage to the cardiovascular system with conduction block and arrhythmia, arterial hypotension and syncope. This treatment can improve the quality of life of AIDS patients, but does not affect the progression of the disease.

Inflammatory demyelinating polyneuropathy

Inflammatory demyelinating polyneuropathy (DIP) develops in the early stages of AIDS and can be a sign of seroconversion (the appearance of HIV antibodies in the patient's blood). The clinical picture is very similar to Guillain-Barré syndrome in people who do not have AIDS. The reason is a violation of immunity and the development of demyelination of peripheral nerves. ZDP can develop at any stage of AIDS, and also have a course with remissions and exacerbations.

The clinic is accompanied by increasing muscle weakness, areflexia, various sensitive and vegetative disorders. Some patients develop respiratory failure. The diagnosis is based on the clinical picture, the study of TSR, HIV disease in the anamnesis. In the treatment, plasmapheresis is used in a volume of 200–250 ml/kg (5 procedures within 2 weeks), intravenous administration of human immunoglobulin at a dose of 0.4 g/kg/day No. 5. Side effects are manifested in the form of myalgias, arthralgias, headaches , dizziness. The prognosis may be positive, after treatment, there may be recovery or residual neurological deficit.

HIV myopathy

Myopathy occurs as a result of AIDS or toxic damage after treatment with zidovudine (AZT). The exact pathogenesis of HIV myopathy has not been clarified. In the clinic, there is symmetrical progressive muscle weakness with an increase in the level of creatine kinase in blood serum. Changes typical for myopathy are registered on EMT — shortening and decreasing the amplitude of motor unit potentials, inclusions in the form of rod-shaped nemaline bodies.

In the treatment, AZT is canceled and corticosteroid therapy in the form of prednisolone is used at a dose of 40–60 mg/day with a gradual dose reduction or

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administration every other day. You can prescribe human immunoglobulin. The forecast is doubtful

Aseptic meningitis

It develops when antibodies to HIV infection appear in the blood. In the clinic, patients suffer from headache, fever, meningeal symptoms, and often - cranial nerve damage, confusion. In the treatment of this disease, only analgesics are used. In SMP there is a moderate lymphocytic pleocytosis, an increased level of immunoglobulins, sometimes oligoclonal antibodies are detected. It is necessary to differentiate aseptic meningitis from neurosyphilis. Cryptococcal infection in AIDS patients Cryptococcal infection is the most common cause of meningitis in AIDS patients (about 10% of patients). In addition to damage to the brain, damage to the lungs, bone marrow, liver, and skin can be observed. The causative agent is transmitted by airborne droplets, penetrates into the central nervous system (CNS) by the hematogenous route.

The clinic is manifested by headache, fever, damage to cranial nerves, convulsive attacks. An increase in the level of cryptococcal antigen is detected in CMP and blood serum. A brain biopsy is required to confirm the diagnosis. Treatment consists in prescribing amphotericin at a dose of 0.6–1.0 mg/kg/day simultaneously with flucytosine at a dose of 25.0–37.5 mg/kg 4 times a day. An alternative is treatment with fluconazole in a dose of 200 ml twice a day. Treatment is long-term (up to 10 months - 1 year). The mortality rate from cryptococcal meningitis is 10–25%. The appearance of mental disorders is considered a bad prognosis.

Central nervous system lymphoma

Primary CNS lymphoma occurs in 2–4% of AIDS patients. The prognosis is poor, most patients die. The clinic is manifested by headache, drowsiness. Hemiparesis, aphasia, ataxia, loss of visual fields may be observed. Leptomeningeal lymphomas with signs of meningitis, meningoencephalitis or meningoradiculitis are detected. MRI helps in diagnosis, sometimes the detection of tumor cells in SMP. Treatment: radiation therapy, and this method is only palliative.

Progressive multifocal leukoencephalopathy (PML)

It occurs in 5% of AIDS patients. There is a subacute, progressive course of the disease in the form of cognitive impairment, loss of visual fields, hemiparesis, ataxia, and speech impairment. Diagnosis: MRI allows visualization of the affected areas. There is no treatment, the prognosis is unfavorable. After diagnosis, patients live no more than 6 months. Therefore, patients with HIV-associated AIDS can have many diseases of the brain and spinal cord, peripheral nervous system, which can develop acutely or gradually, but necessarily with progress by There is no specific therapy, the prognosis in most cases is unfavorable, some patients die after 6 months from the moment of diagnosis.

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Such patients come to the hospital more often with a diagnosis of meningitis, meningoencephalitis, polyneuropathy, Guillain-Barré syndrome, brain tumors. And only anamnesis, clinic, additional research methods (MRI, CMR, biopsy, laboratory data) help to determine the diagnosis.

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