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OPPORTUNISTIC DISEASES OF THE NERVOUS SYSTEM IN HIV INFECTION

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ABSTRACT

This article is devoted to the study of the clinical manifestations of lesions of the central nervous system caused by various reasons against the background of the progression of HIV infection. The concentration of p24 antigen and antip24 antibodies in the cerebrospinal fluid and serum varies in parallel, but the concentration of p24 in the cerebrospinal fluid is usually higher. The tumor spreads perivascular, and the clinical picture depends on the location and volume of the tumor. In the treatment of patients with HIV infection with the development of clinical lesions. Patients with a positive culture of HIV from cerebrospinal fluid have both anti-IV antibodies in the cerebrospinal fluid and oligoclonal bands. In patients with AIDS, the synthesis of antibodies in the cerebrospinal fluid is noticeably lower than in HIV-infected people without AIDS. The tumor spreads perivascular, and the clinical picture depends on the location and volume of the tumor. In the treatment of patients with HIV infection with the development of clinical lesions.

KEYWORDS: *Multifocal Encephalopathy, Polyradiculoneuritis, Magnetic Resonance Imaging, Cerebral Toxoplasmosis, Oligoclonal Antibodies, Cryptococcal Meningitis, Cerebrospinal Fluid.*

INTRODUCTION

The most important of this group of diseases are progressive multifocal encephalopathy, cerebral toxoplasmosis, cryptococcal meningitis, encephalitis and polyradiculoneuritis caused by cytomegalovirus and viruses of the genus Herpesvirus, tuberculosis with brain damage, primary lymphoma of the central nervous system. The diagnosis of opportunistic infection is often verified only retrospectively when responding to specific therapy; suspicion may arise from the analysis of nonspecific clinical symptoms, computed tomography and magnetic resonance imaging data, and based on serological studies or biopsy data.

Approximately 40% of HIV-infected people have altered cerebrospinal fluid, usually in the form of mild pleocytosis (5-50 cells / mm3), increased amounts of protein (500-1000 mg / L) and normal glucose concentration. These changes are not specific. Half of clinically healthy HIV-infected patients have pleocytosis or an increased amount of protein in 20% of the cerebrospinal fluid gives rise to HIV in tissue cultures, often in high titers. Later, pleocytosis decreases, while the amount of protein may increase, decrease, or be unchanged. As in peripheral blood, the cerebrospinal fluid CD4: CD8 ratio is low, especially in late infection. The titer of the virus in the cerebrospinal fluid in the late stage also decreases. These changes in the cerebrospinal fluid are moderately expressed and not constant; therefore, based on them, it is difficult to predict the course of the disease and the effectiveness of therapy.

Anti HIV is usually detected in the cerebrospinal fluid in a high titer. Comparison of antibody titers in blood and cerebrospinal fluid indicates that antibodies can be synthesized in the central nervous system. Antibodies to HIV in the cerebrospinal fluid belong to the IgG class, but in some patients it was possible to find antibodies of the IgA and IgM classes. The synthesis of antibodies in the central nervous system begins early, immediately after infection of the meninges. Oligoclonal antibodies in the cerebrospinal fluid can also be detected, they correspond to HIV epitopes and have a different migration ability from serum antibodies. Pleocytosis and protein concentration correlate poorly with anti-HBV antibodies in the cerebrospinal fluid and the presence and number of oligoclonal bands. Patients with a positive culture of HIV from cerebrospinal fluid have both anti-IV antibodies in the cerebrospinal fluid and oligoclonal bands. In patients with AIDS, the synthesis of antibodies in the cerebrospinal fluid is noticeably lower than in HIV-infected people without AIDS. The concentration of p24 antigen and antip24 antibodies in the cerebrospinal fluid and serum varies in parallel, but the concentration of p24 in the cerebrospinal fluid is usually higher. The concentration of p24 is maximal in the AIDS-dementia complex, but usually the concentration of antigens and antibodies correlates poorly with the severity of clinical symptoms and the effectiveness of the therapy.

In the clinical picture, a characteristic series of symptom complexes can be distinguished: meningism, pyramidal insufficiency, cerebellar ataxia, convulsive syndrome, AIDS-dementia complex, symptom complex characteristic of encephalitis, meningitis. Clinical observations show that in the early stages of HIV infection, the most common are reactive neurotic states and manifestations of asthenovegetative syndrome. Patients have a variety of neurotic disorders, as well as increased fatigue, absent-mindedness, forgetfulness, mood deterioration, narrowing of the range of interests, sleep disorders, various phobias, autonomic ability. In the later stages of the disease, damage to the nervous system comes to the fore, mainly due to opportunistic infections.

With progressive multifocal leukoencephalopathy, there are clinical manifestations of multifocal lesions of the white matter of the brain in the form of hemiparesis and hemigipesthesias, hemianopsia, static and dynamic ataxia, which may be accompanied by a decrease in intelligence, seizures. Symptoms slowly and steadily progress to complete immobility of patients. This encephalopathy is caused by the JC papovavirus, which acts simultaneously with HIV. In addition to foci of demyelination, the identification of glial cells with characteristic inclusions around areas of myelin destruction is pathognomonic. There is no effective treatment for this disease. The prognosis is poor, since the maximum life expectancy after the onset of the first symptoms does not exceed 2 months.

Cerebral toxoplasmosis is a consequence of the reactivation of a latent brain infection by the intracellular parasite Toxoplasma gondii. The clinical picture is due to the localization and activity of the inflammatory process. There are no specific clinical symptoms of this encephalitis. On tomography, multiple bilateral annular foci are often noted, although these changes are also not specific. An accurate diagnosis can be made on the basis of data on the detection of this pathogen by various methods in biopsy samples. In some cases, the diagnosis is confirmed indirectly if the patient's condition improves after the appointment of specific drugs (pyrimethamine, sulfadiazine, etc.).

Cryptococcosis and other systemic mycoses kill up to 10% of HIV-infected worldwide. Cryptococcosis often occurs in the form of meningitis, pneumonia and disseminated infection develop less often.

Cryptococcal meningitis is the most common systemic mycosis in HIV-infected people. Clinical manifestations include headache, fever, neck stiffness, cranial nerve damage, impaired consciousness up to coma. However, symptoms of meningitis, including fever and a stiff neck, are often absent. Without treatment, the life expectancy of patients is less than a month.

Diagnostics. The diagnosis of cryptococcosis is relatively easy. The cerebrospinal fluid is centrifuged, the resulting sediment is examined under a microscope after adding a drop of ink. The preparation reveals yeast cells covered with a thick capsule. Other diagnostic methods are the isolation of a culture of cryptococcus from the cerebrospinal fluid and examination of plasma or cerebrospinal fluid for cryptococcal antigen. Severe multifocal polyradiculoneuropathy caused by cytomegalovirus is virtually untreatable.

This syndrome is usually accompanied by other manifestations of infection: pneumonia, colitis, rhinitis, etc.

Reactivation of latent infection caused by Micobacterium tuberculosis leads to the development of tuberculous meningitis, brain abscesses.

The brain abscess itself is a local accumulation of pus in the substance of the brain. The clinical presentation may include headache, lethargy, fever, and focal neurologic symptoms. Diagnosisisbycontrast MRI orcomputedtomography.Treatment with antibiotics and, as usual, with stereotaxic aspiration under the guidance of computed tomography or surgical drainage. If a brain abscess is suspected, diffusion spectral MRI or (if this is not possible) computed tomography with contrast enhancement is necessary. In some cases, it may be necessary to conduct computed tomography-directed aspiration with taking material for bacteriological examination or excision of the abscess area, it is also possible to combine these approaches. A fully formed abscess is visualized as an area with edema and an increase in the signal along the periphery, which is sometimes difficult to differentiate from a brain tumor and, rarely, an infraction zone.

Bacteriological examination of pus sucked from the abscess can make targeted antibiotic therapy of the abscess possible. However, antibiotics should not be withdrawn until culture results are available.

Severe diffuse encephalitis in AIDS patients can be caused by Herpes simplex and Varicella zoster viruses. In 5% of AIDS patients, primary lymphoma of the central nervous system (mainly

B-type, in the genesis of which infection with the Epstein-Barr virus is of great importance) and Kaposi's sarcoma, sometimes leading to the development of intracerebral hemorrhages, can be detected. Primary central nervous system lymphoma is an AIDS-specific manifestation. Usually atypical lymphocytes proliferate. The tumor spreads perivascular, and the clinical picture depends on the location and volume of the tumor. In the treatment of patients with HIV infection with the development of clinical lesions. The central nervous system must first of all exclude cerebral toxoplasmosis and, without waiting for the test results, start therapy, biseptol (ex juvantibus) 10 mg / kg for trimethoprim in 2 doses i / v, drip or oral.

It is believed that a clear clinical effect can be expected after 8-10 days from the start of therapy, and positive changes on MRI as early as 2 weeks. The duration of treatment is at least 6 weeks (before elimination of at least 75% of foci on MRI of the brain). But often this does not happen.

- Prevention of recurrence of toxoplasmosis - half doses of the treatment course (usually 960 mg 2 times a day for 6 months after reaching a CD4 level of more than 200 in μl). But if perifocal edema and multiple lesions persist on control MRI, a longer prophylactic dose of the drug is possible.

- With the development of toxic reactions to biseptol - it is possible to use spiramycin, fancidar, combinations of other drugs.

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