CHARACTERISTICS OF CLINICAL DIAGNOSIS OF NERVOUS SYSTEM INVOLVEMENT IN PATIENTS WITH HIV INFECTION

Mo’minov A.O
Usmanova E.M
Andijan State Medical Institute

Resume: *Among the observed patients, the predominant route of HIV transmission was through injection drug use. The most common pathogens affecting the central nervous system (CNS) in HIV-positive patients were the Epstein-Barr virus, Toxoplasma, and Mycobacterium tuberculosis. The disease primarily developed in young individuals, regardless of gender. CNS infections in HIV-positive patients progress gradually and manifest clinically against a background of severe immunodeficiency.*

Key words: *HIV-infected patients, central nervous system, PCR, meningeal signs, CD4 cells.*

Relevance. HIV infection is one of the most common and dangerous human viral infections. Among the most pressing problems associated with this disease, the most important place is occupied by lesions of the central nervous system (CNS), primarily the brain. The fact that various reasons can underlie these defeats is of significant importance:

1) direct damage to brain tissue by the human immunodeficiency virus;

2) connection of brain damage with cytokines produced by infected perivascular macrophages;

3) exposure to pathogens of a number of secondary infections;

4) neoplasms, primarily isolated cellular lymphomas [1].

Despite the existence of antiretroviral therapy, HIV infection is an incurable disease, one of the most important problems of which is the damage to the nervous system, occurring in 50-80% of cases [3, 5, 8].

Primarily, damage occurs in the 3rd and 4th stages of the disease [2, 7], although there are sources reporting damage in the early stage [4]. One of the formidable complications of CNS damage is acute encephalitis, which in most cases occurs in the first months of disease development with clinical manifestations in the form of fever, general malaise, mood changes, epileptic seizures, changes in consciousness.

In addition to the above-mentioned symptoms, lesions of cranial nerves also occur in meningoencephalitis, occurring in 5-10% of cases. The most severe damage occurs in the V, VII, and VIII cranial nerve pairs [4, 8].

Materials and methods. We examined 73 HIV-infected patients.

The etiological factor was determined based on cerebrospinal fluid analysis using PCR to detect DNA fragments of herpes viruses types 1, 2, 3, and 6, cytomegalovirus, Epstein-Barr virus (EBV), and Toxoplasma. Microscopic and microbiological

laboratory methods were used to confirm the tuberculous etiology of central nervous system (CNS) lesions.

Results obtained and their discussion.

The study included 73 HIV-infected patients with central nervous system (CNS) damage, with a mean age of 35,7±1.3 years, comprising 54,9% men and 45,1% women.

The etiological factor of CNS damage was established in 58,5% HIV-infected patients. In 41,4% patients, the etiology remained unidentified. Examination of the disease history revealed that the illness began gradually in all patients.

The average time from onset of symptoms to hospital admission was 42 days. The majority of patients, 60,0%, were admitted to the hospital in moderate condition, while 35,0% patients were admitted in severe condition. Impaired consciousness was observed in 30,0% patients.

The severity of the disease was attributed to the degree of intoxication and immunodeficiency, as well as the intensity of neurological symptoms and the development of complications such as cerebral edema and swelling, which were the immediate causes of death.

As a consequence of brainstem damage, disorders of cranial nerves (II-VII, IX, XII) were observed. Visual acuity decreased in 10% of patients, while bulbar syndrome was observed in 17,5% of patients.

Vestibulo-ataxic syndrome was more common and manifested as dizziness in 82,5% of cases, unsteady gait in 60.0%, and unsteadiness in the Romberg position in 51,3% of cases.

Primary neuroAIDS caused by HIV itself can manifest in various clinical forms: AIDS dementia (HIV encephalopathy), meningitis or meningoencephalitis, vascular neuroAIDS, vacuolar myelopathies of ascending or transverse type, symmetrical sensory distal polyneuropathy, chronic inflammatory demyelinating polyneuropathy, acute inflammatory demyelinating polyneuropathy (AIDP) of the Guillain-Barré syndrome type, encephalomyelopolyneuropathy, and ALS-like syndrome (ALS-amyotrophic lateral sclerosis).

Radiological examination of patients in the 3rd group revealed specific lung inflammation in 75% of patients, suggesting tuberculous lesions of the CNS. Immune status assessment showed that in patients of all groups, the level of CD4+ cells did not exceed 100 per 1 μl. The average number of CD4+ cells in the 1st group was 49 per 1 μl, in the 2nd group - 55 per 1 μl, and in the 3rd group - 91 per 1 μl.

Conclusions. The clinical presentation of central nervous system (CNS) infections in HIV-positive patients has distinct features depending on the etiology.

A thorough examination of the patient's neurological status and the combination of specific symptoms will help the physician to hypothesize the etiology of CNS involvement and initiate early targeted therapy.

Clinical analysis and PCR testing of cerebrospinal fluid play a crucial diagnostic role in confirming the etiology of CNS damage.

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