



## Anxiety and Depression in Patients with HIV Encephalopathy

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ABSTRACT

This article examines the level of anxiety and the degree of depression in patients with HIV-associated encephalopathy. The patients were examined using the State-Trait Anxiety Inventory (STAI) and the Hamilton Depression Scale .

**Keywords:**

HIV infection, HIV - encephalopathy, anxiety, depression, quality of life.

**Introduction:** HIV encephalopathy is a serious complication of the human immunodeficiency virus (HIV) resulting from brain inflammation. HIV affects many body systems, including the immune and central nervous systems. When the virus enters the brain, various mental and intellectual disorders occur. When HIV infection causes swelling of the brain, it is called HIV encephalopathy. Other names for this disease are HIV-associated complex dementia and AIDS dementia. This condition can affect motor functions and cognitive abilities and lead to dementia. HIV encephalopathy cannot be cured, but it can be slowed down or controlled with treatments such as antiretroviral therapy.

Direct damage of central nervous system tissue due to HIV combined with activated immune responses to infection has been implicated in causing the neurocognitive decline associated with HIV encephalitis. HIV infection results in rapid seeding of the virus in various body premises, including CNS tissues, which is usually asymptomatic. Following infection with HIV, monocytes circulating in the blood migrate to the brain after traversing the blood-brain barrier, thus enabling HIV to gain access to the central nervous system. The entry of HIV into brain tissue through brain endothelium is

believed to be aided by the induction of vascular cell adhesion molecule 1 (VCAM-1) and E-selectin. Though the direct impact of HIV on neurocognitive dysfunction after further development of distinct genetic sequences has been studied, the more significant impact has been attributed to the indirect immune pathways triggered after viral entry into brain tissue.[7]

The release of monocyte-derived cytokines such as IL-1, TNF- $\alpha$ , TGF- $\beta$ , which trigger neurotoxic changes, is believed to be the more damaging entity in the pathophysiology of HIV encephalopathy.[8] Increased levels of monocytic chemotactic protein-1 in cerebrospinal fluid (CSF) have been shown to correlate with the severity of HIV encephalopathy.

The compartmentalization of HIV infection in the CNS also plays a particularly important role in developing HIV encephalitis. After the initial development of the virus from the circulating CD4 cells, in later stages of infection, CSF viral replication occurs independently of plasma viral replication. A change in the tropism of the CNS viruses in the later stages of the infection also worsens the severity of the illness.

Several factors determine the neurological deterioration in HIV encephalitis. The

production of neurotoxic HIV proteins, uncontrolled viral replication in brain tissue, and immune activation are underlying factors controlling the rate of neurological decline.[9] HIV infection and its spread is one of the main problems of modern medicine in the world. Already in the early stages of systemic HIV infection, the brain and soft membranes are exposed to the virus, which is accompanied by an immune response within the brain [1,2] . It is emphasized that both viral aggression and immune reactions are metastatic, since they represent the translocation of virus-infected body cells and immune cells from the systemic circulation to the brain. The authors identify two factors for the consistent development of the clinical picture of damage to the nervous system in HIV infection [3] . The first factor is the effectiveness of immune protection. In this case, this is manifested by the degree of suppression of virus replication within the brain (AIDS dementia and HIV encephalitis do not develop in all patients with high viremia ). The second factor is the emergence of macrophage -tropic genetic variants of the virus [4,5] .

**Purpose of the study.** The aim of our study is to determine the degree of anxiety and depression in patients with HIV-encephalopathy, depending on the duration of the disease.

**Materials and research methods.** For the study, 53 patients were randomly selected who were treated at the Fergana branch of the Republican AIDS Center. Among them, 33 men (62.3%) and 20 women (37.7%), the average age of patients is  $31.7 \pm 1.1$  years. To study anxiety, a battery of tests was chosen - Spielberg - Khanin, and to study the level of depression, the Hamilton scale.

**Research results.** As a result of the study, the Spielberg-Khanin test determined increased personal anxiety compared to reactive anxiety. The highest anxiety is determined in the first 2 years from the date of diagnosis. It is described in more detail in table No. 1

Table #1

	Reactive	Personal
0-2 years	$38.2 \pm 4.82$	$56.5 \pm 4.76$
2-5 years	$28.9 \pm 2.71$	$49.3 \pm 2.31$
5-7 years	$30.8 \pm 2.92$	$53.6 \pm 2.51$
7 years or more	$19.6 \pm 5.13^*$	$34.5 \pm 8.4^*$

In the study of depression using the Hamilton scale , the most pronounced depressive state was observed in the first 2 years of the disease with gradual stabilization to the level of mild depression. In more detail, the results are disclosed in table No. 2

Table number 2

	Hamilton scale
0-2 years	$16.2 \pm 1.15$
2-5 years	$12.4 \pm 0.71$
5-7 years	$11.9 \pm 0.92$
7 years or more	$12.2 \pm 0.13$

**Conclusions.** Studies of anxiety and the severity of depression showed the highest rates in the initial stage of the disease. When using enhanced psychological support , as well as the use of antidepressants with anxiolytic activity, it can stabilize the patient's psychological state and improve the patient's quality of life.

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