



HIV MENTAL HEALTH TREATMENT ISSUES

HIV and Cognitive Disorders

Still a problem for many HIV+ persons.

The brain is one of HIV's main targets. The virus most likely crosses the blood-brain barrier into the central nervous system (CNS) soon after infection. This means there are significant nerve and brain impacts in most HIV+ people and a wide range of clinical neurological symptoms. HIV-related clinical and neuropsychological disorders are more common in drug abusers than among other populations.

The clinical manifestations of CNS disorders in HIV disease include depression and all degrees of cognitive impairment. Neurocognitive disorders are most common in late-stage HIV disease (AIDS), unlike anxiety disorders (frequently seen just after HIV diagnosis) and depressive disorders (frequently seen when HIV-related symptoms appear).

HIV-Associated Neurocognitive Disorder (HAND) is still common even among individuals receiving highly active antiretroviral therapy (HAART). HAND risk is correlated with the nadir (low point) of CD4 cell count and an HIV viral load in the cerebrospinal fluid at least as high as plasma viral loads. Age, methamphetamine use, coinfection with hepatitis (A, B, or C), and a family history of dementia also increase an HIV+ person's risk for neurocognitive disorders.

What cognitive disorders are linked to HIV?

The American Academy of Neurology recognizes two clinical neurocognitive disorders diagnosed by use of standard psychological tests: **Mild Neurocognitive Disorders (MND)** and **HIV-Associated Dementia (HAD)**. An **MND** diagnosis requires mild neurocognitive impairment in at least two domains of cognitive performance and, at most, a minor functional impairment that isn't severe enough for a HAD diagnosis. A **HAD** diagnosis requires cognitive impairment in two or more domains, at least a moderate level of functional impairment due to cognitive symptoms, a lack of delirium, and no evidence of another explanation for the symptoms.

Asymptomatic Neurocognitive Impairment (ANI)

ANI occurs without any related decrease in function. High rates of ANI have been found in HIV+ young adults (67%) who were infected perinatally, compared with older HIV+ persons (19%). Other studies have found as many as 1 in 3 HIV+ persons taking HAART had ANI, but again they were mostly under 40 years old. Because of recent challenges in the diagnosis of ANI a cautious approach for monitoring patients for disease progression is recommended.

HIV-Associated Dementia (HAD)

Before HAART was available, HIV-associated dementia was a common AIDS-related complication and cause of death. With HAART the incidence of HAD has fallen, although its prevalence has actually increased because HIV+ people are living longer. Even today, HAD affects 10-20% of people living with HIV.

HAD's exact causes are unclear, although it corresponds most closely to inflammation in the brain rather than with viral load or HIV encephalitis. Neuroinflammation has long been recognized as a common pathological finding in HIV+ individuals and has been linked with CNS dysfunction.

Symptoms and signs of HAD include tremor, gait ataxia, loss of fine motor movement, mental slowing, forgetfulness, poor concentration, and behavioral abnormalities.

Risks for HAD include older age, decreased body mass, family history of dementia, and persistent physical symptoms of HIV infection. As many as 15% of those with advanced HIV disease (AIDS) are affected by HAD, severely impairing their daily functioning.

Potential cognitive changes from HAART medications

Some potential side-effects of HAART and of HIV infection itself may overlap, particularly in people with advanced HIV disease (AIDS). Either HIV or HAART can cause neurologic complications including changes in cognition and dementia.

The level of neuroinflammation remains unexpectedly high even in HIV+ people treated with HAART. HAART-treated individuals also may have the nerve-destroying proteins normally associated with Alzheimer's in both the brain and cerebrospinal fluid.

In some cases, CNS complications—including psychiatric syndromes, delirium, seizures, and cognitive impairment—may be the result of HAART drugs penetrating the blood-brain barrier. Zidovudine and efavirenz, both of which are used to treat patients with CNS complications because they are able to penetrate the CNS, are themselves associated with potentially significant neuropsychiatric complications.



References

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About this Fact Sheet

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