

Assessment and Diagnosis of HIV-associated Dementia

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ABSTRACT

HIV infection has long been known to result in dementia and other forms of neurocognitive deficits. The rate of HIV-associated dementia is decreasing, while mild forms of neurological impairments increase. Treatment of HIV infection has advanced, and patients are living longer, and are thus at a greater risk of cognitive decline. The HIV aging cohort is susceptible to neurocognitive impairment from other medical conditions that have a compounding effect on cognitive decline. The diagnosis of HIV-associated neurocognitive disorders involves identifying neurological dysfunction and then determining that HIV is the most probable cause. Implications for practice include early control of HIV replication and treatment of comorbid diseases.

Keywords: HIV-dementia (HAD), HIV-associated neurocognitive disorder (HAND), AIDS dementia complex, combined antiretrovirals (cART), HIV treatment, hepatitis C

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INTRODUCTION

Dementia and other forms of neurocognitive deficits have been reported among people living with HIV (PLWH) since the beginning of the HIV epidemic.¹ With over 1.2 million PLWH in the United States² there is concern that half of them may be at risk of developing cognitive deficits.³ Management of HIV infection has significantly advanced, and patients who adhere to medications are living with the chronic infection for longer into old age, thus putting them at greater risk of cognitive decline.⁴ Despite the progress in HIV treatment, patients on highly active combined antiretrovirals (cART) are still presenting with varying degrees of neurocognitive deficits from early stage HIV seroconversion to late-stage disease.^{5–7} It is vital for Nurse Practitioners (NPs) to diagnose symptomatic neurocognitive impairment in PLWH because it is associated with non-adherence to antiretrovirals, lower quality of life, and increased mortality.⁸ This article aims to 1) review the literature on HIV-associated neurocognitive disorders; 2) propose clinical recommendations for NPs concerning best clinical practice; and 3) explore the gaps in research

on the neurocognitive decline associated with HIV infection.

Neurocognitive decline associated with HIV was originally known as AIDS dementia complex and was later changed by a National Institutes of Health working group to HIV-associated dementia (HAD).⁹ With the introduction of cART in 1996–1997, mild cognitive and motor disorders that did not meet criteria for dementia became apparent, and the need to better define this spectrum led to the term HIV-associated neurocognitive disorders (HAND).⁹

Since cART, several studies in developed nations have reported a decrease in HIV dementia but an increase in the prevalence of milder forms of cognitive impairment.^{3,10,11} Milder forms of HAND include asymptomatic neurocognitive impairment (ANI) and mild cognitive disorder. ANI is the most widespread type of HAND, accounting for more than half of cases. In ANI, there is the presence of definite impairment on neurocognitive testing, but there is no apparent effect on daily functioning.¹² The rise in ANI and mild cognitive disorder remains a mystery, but researchers theorize that it could be related to the aging of the central nervous system (CNS), drug toxicity, or prolonged immune

activation.^{3,13} Higher prevalence is concerning because there is growing evidence that patients with ANI have a higher risk of deteriorating to severe forms of HAND.¹⁴

It appears the use of cART has altered the trajectory of HAND. Patients presenting with HAND may have different risk factors from pre-cART.⁴ Uncontrolled HIV infection reflected by nadir CD4 count was associated with dementia in the pre-cART era, but current evidence shows a subset of patients develop HAND despite achieving undetectable plasma HIV RNA.⁸ Also, some studies show that some PLWH have inconsistent neurocognitive symptoms that fluctuate from normal to abnormal; this is different from other neurodegenerative diseases such as Alzheimer's.⁹

The pathogenesis of HAND continues to be poorly understood. In clinical practice, HIV is considered a possible source of neurocognitive impairment, but there are no studies that definitively delineate other clinical and psychological factors that may contribute to HAND.¹⁵ In the age of cART, PLWH are living longer and have comorbidities such as cardiovascular disease, metabolic disease, and hepatitis coinfections that have been shown to contribute to neurocognitive damage.^{16–18} It is also well established that aging is a major risk factor for dementia. With 19% of the HIV population over the age of 55, NPs should expect more cases of HAD.¹⁸ Measurable markers of cerebrospinal fluid (CSF) inflammation in early neuro-invasions indicate that the CNS is a target organ by HIV.³ Several theories have been hypothesized to explain the neurovirulence of HIV. For example, the HIV-infected monocytes cross the brain barrier, resulting in the release of cytokines and viral products that interrupt the blood brain barrier and are detrimental to neuronal pathways.¹¹ Another basis of neuronal damage could result from chronic inflammation and ongoing HIV replication that persists despite peripheral HIV suppression.^{3,8}

RISK FACTORS

Risk factors for the development of HAND are not well defined. Though any HIV-infected individual could be at risk of developing neurocognitive deficits, the role of host factors such as genetic predisposition, aging, cardiovascular and metabolic factors, and

hepatitis C comorbidities remain unascertained.¹⁹ Some studies have associated depressed nadir CD4 cell count with HAND,^{8,19} other researchers have not been able to replicate this finding that links neurological impairment and virology status.²⁰ Data remains sparse regarding the role of aging in the development of HAND. One study found that PLWH older than 50 years were 3 times as likely to present with neurocognitive impairment compared with 20–39-year-old HIV patients.^{4,20}

CLINICAL PRESENTATION

The severe form of HAND typically seems to occur in patients with advanced HIV infections, low CD4 cell counts, and high peripheral viral levels. Subcortical dysfunction fundamentally characterizes early stage HAD.⁹ Clinical features of subcortical impairment include attention—concentration impairment, depressive symptoms, and impaired psychomotor speed and precision. As the disease advances, symptoms typical to cortical dementias such as aphasia, agnosia, and apraxia are manifested.^{8,9} The clinical neurological features of HAND are described in [Table 1](#).

The severe form of HAND was prevalent before the cART era. In developed countries, it is rare for successfully treated HIV individuals to present with HIV dementia. However, the high prevalence of milder forms of cognitive impairments suggests that

Table 1. Clinical Features of HAND

| | |
|-------------------|---------------------------|
| Early stage signs | Short-term memory loss |
| | Poor concentration |
| | Apathy |
| | Poor comprehension |
| | Depression |
| | Tremor and poor dexterity |
| | Hyperreflexia |
| | Unsteady gait |
| | Impaired ocular movements |
| Late-stage signs | Global dementia |
| | Neuropathy |
| | Myelopathy |
| | Frontal release signs |
| | Parkinsonism |
| | Ataxia |
| Seizures | |

Abbreviation: HAND, HIV-associated neurocognitive disorders.
Data from McArthur et al.¹¹

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